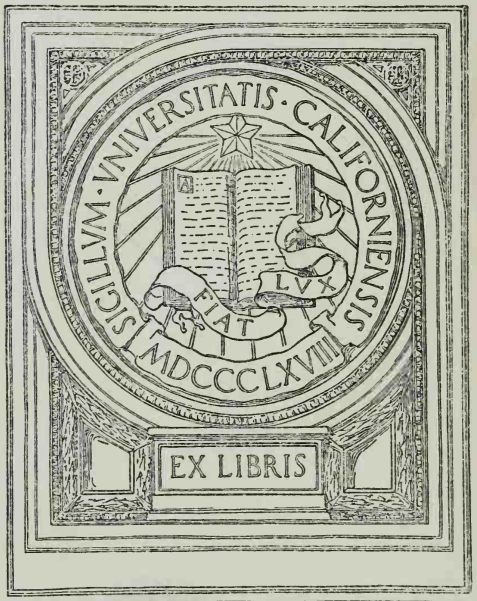
SURGICAL ASPECTS OF TYPHOID AND PARATYPHOID FEVERS

A. E. WEBB-JOHNSON

OXFORD MEDICAL PUBLICATIONS

MEDICAL SCHOOL
LIBRARY



GIFT OF THE LATE
DR. ESTHER ROSENCRANTZ

To Professor Rosencrantz
with best wishes from the Author
London
9/8/23.

SURGICAL ASPECTS OF TYPHOID AND PARATYPHOID FEVERS

PUBLISHED BY THE JOINT COMMITTEE OF
HENRY FROWDE AND HODDER & STOUGHTON
AT THE OXFORD PRESS WAREHOUSE
FALCON SQUARE, LONDON, E.C.1

SURGICAL ASPECTS OF TYPHOID AND PARA-TYPHOID FEVERS

FOUNDED ON THE HUNTERIAN LECTURE FOR 1917—AMPLIFIED AND REVISED

BY

A. E. WEBB-JOHNSON, D.S.O.

M.B., Ch.B. (Vict.), F.R.C.S. (Eng.); Temporary Colonel, Army Medical Service; Consulting Surgeon, British Expeditionary Force, France
Hunterian Professor of Surgery, Royal College of Surgeons of England
Assistant Surgeon to the Middlesex Hospital.

With Foreword by

LIEUT.-GENERAL T. H. GOODWIN

C.B., C.M.G., D.S.O.

Director-General, Army Medical Service.

LONDON

HENRY FROWDE HODDER & STOUGHTON

OXFORD UNIVERSITY PRESS WARWICK SQUARE, E.C.

1919

PRINTED IN GREAT BRITAIN
BY HAZELL, WATSON AND VINEY, LD.,
LONDON AND AYLESBURY.

RC
187
W36s
1919

TO

THE ROYAL ARMY MEDICAL CORPS

82550

FOREWORD

By the Director-General, Army Medical Service

It is probable that a more profound influence has been exercised on armies in the field by typhoid and paratyphoid fevers than by any other group of diseases.

When we consider the history of previous campaigns we may, I think, congratulate ourselves that the importance of this group of diseases has been so fully recognised and so adequately dealt with, both prophylactically and otherwise, during the present war.

These diseases have usually been regarded as falling to such a large extent within the province of the physician that the surgical complications and sequels have perhaps not received attention to the degree which they merited. With the exception of the valuable work of Professor Keen, of Philadelphia, written in the year 1898, comparatively little literature has been contributed on the subject.

Colonel Webb-Johnson's book is not only intensely interesting historically, but is also of extreme value to the surgeon.

Our knowledge on this subject has greatly expanded during the present war, more especially regarding the paratyphoid fevers, with respect to which much valuable work has been carried out.

The author of this book had unique opportunities for closely observing a large series of cases in this group of diseases, and he utilised these opportunities to the best possible advantage.

The "carrier" problem is one of immense interest and importance, not only to the Army but also to the civil community, and the views regarding this and the rôle played by the spleen are ably set forward.

This and many other points of interest are adequately dealt with in these pages.

In placing the results of his study and experience before the profession, Colonel Webb-Johnson has conferred a benefit on surgeons which they will sincerely appreciate.

T. H. Goodwin, D.G.

AUTHOR'S PREFACE

When asked to write a book on the Surgical Aspects of the Typhoid Group of fevers, I willingly agreed, as I had already discussed them in the Hunterian Lecture in 1917 (*The Lancet*, December 1st, 1917), but I recognised that it would be impossible to deal exhaustively with the subject while working under war conditions. At the same time, I felt that the classical work of Professor W. W. Keen on *The Surgical Complications and Sequels of Typhoid Fever* (W. B. Saunders & Co., 1898) was still a standard authority, and that the subject had been dealt with so thoroughly by this Nestor of English-speaking surgeons that even twenty years after the publication of his book all that was called for was a consideration of new problems, a review of advances in our knowledge, with an outline of our present conception of these fevers, their complications and sequels, and a description of modern surgical practice with regard to them. To produce such a review required the ready help and co-operation of my colleagues at the hospital in France, on the work of which it would mainly be based. I knew from experience that this help would be forthcoming in full and generous measure, an experience very different from that of

John Hunter, who, when serving with the British Expedition to Belleisle, wrote home to his brother, "My fellow-creatures of the hospital are a damn'd disagreeable set."

I am particularly indebted to Major-General Sir Bertrand Dawson, G.C.V.O., C.B., for much friendly assistance, to Colonel R. Davies-Colley, C.M.G., who left all his material at my disposal when he volunteered for service in Mesopotamia, and to Colonel C. R. Evans, D.S.O., and Lieut.-Colonel J. R. Harper, T.D., the officers commanding the hospital. Professor Dreyer worked at the hospital for many months, and in charge of the laboratory we have had Colonel H. W. Grattan, Captain L. Wood, and Captain H. Marrian Perry (Assistant Adviser in Pathology to the B.E.F.), and to all of them I am indebted for help and suggestions. To Colonel A. Fullerton, C.M.G., and my colleagues in the wards I have incurred many obligations for here a little and there a little. To them I wish to express my gratitude, and also to Lieut.-Colonel R. J. C. Thompson, C.M.G., D.S.O., for help in correcting the proofs.

Working under war conditions, I have not been able to refer to much material that I would otherwise have used, and perhaps I need offer no apology for the few references to the literature of the subject.

A. E. Webb-Johnson.

France, 1918.

CONTENTS

CHAPTER I

PAGE

HISTORICAL SKETCH 19

CHAPTER II

INTRODUCTORY 43

CHAPTER III

GENERAL REVIEW OF SURGICAL COMPLICATIONS 50

CHAPTER IV

THE ALIMENTARY TRACT 59

CHAPTER V

THE SPLEEN 83

CHAPTER VI

THE LIVER, BILIARY PASSAGES, AND PANCREAS 88

CHAPTER VII

THE CARDIO-VASCULAR SYSTEM . . . 103

CHAPTER VIII

PAGE

THE URINARY SYSTEM 115

CHAPTER IX

THE GENITAL ORGANS 124

CHAPTER X

PAROTITIS 127

CHAPTER XI

THE RESPIRATORY SYSTEM 131

CHAPTER XII

JOINT COMPLICATIONS 137

CHAPTER XIII

THE MUSCULAR SYSTEM 146

CHAPTER XIV

BONE COMPLICATIONS 149

CHAPTER XV

MISCELLANEOUS ABSCESSES. EYE AND EAR COMPLICATIONS 160

CHAPTER XVI

SURGICAL ASPECTS OF THE CARRIER PROBLEM 167

INDEX 177

LIST OF ILLUSTRATIONS

FIG.		PAGE
1.	Thomas Willis	18
2.	Henry, Prince of Wales	20
3.	Sir Theodore de Mayerne	22
4.	Sir William Jenner	24
5.	Dr. Charles Murchison	26
6.	Dr. William Cayley	27
7.	John Hunter	28
8.	Portion of ileum showing great enlargement of a Peyer's patch	30
9.	Portion of ileum showing enlargement of several small Peyer's patches	30
10.	Portion of ileum exhibiting an advanced stage of typhoid ulceration	32
11.	Cæcum, last part of ileum and appendix in which are several small deep typhoid ulcers nearly healed	32
12.	Portion of colon showing ulcers partly healed	33
13.	Portion of ileum showing two Peyer's patches with hyperplasia and early necrosis	33
14.	Matthew Baillie	34
15.	William Clift	36

FIG. PAGE

16. Sir James Paget 38

17. Professor W. W. Keen 40

18. *Paratyphoid B:* Lower portion of ileum showing numerous ulcers, from which the sloughs have completely separated 44

19. *Paratyphoid B:* A Peyer's patch in the ileum, showing hyperæmia and early necrosis . . . 44

20. *Paratyphoid B:* Ileum and cæcum. The ulcers in the ileum are deep and have separated in part. The cæcum is riddled by small ulcers, some of which have coalesced 45

21. Statue of Jenner in Boulogne 56

22. Portion of ileum in which there is a small deep typhoid ulcer protected on the external surface by a peritoneal adhesion 66

23. *Paratyphoid A:* Ulcer of the appendix with a large perforation 77

24. Enlargement of superficial veins to compensate for obstruction of the external iliac vein . . . 108

25. Patient who died of septicæmia with erysipelas and ulceration of the larynx 134

26. Radiograph of typhoid osteo-periostitis of tibia (Knox) 152

PLATE I. Spleen. *Paratyphoid B:* The spleen shows a large abscess at its lower pole, and along the anterior margin a small cavity has been opened. Between these two cavities the spleen is mottled yellow and blue grey, and indicates an underlying third abscess. The wall of the lower abscess is adherent to the colon . . . . . *facing page* 85

PLATE II. Gall-bladder and gall-stone. *Paratyphoid B:* Acute cholecystitis with abscesses in the wall of the gall-bladder . *facing page* 99

HISTORICAL SKETCH

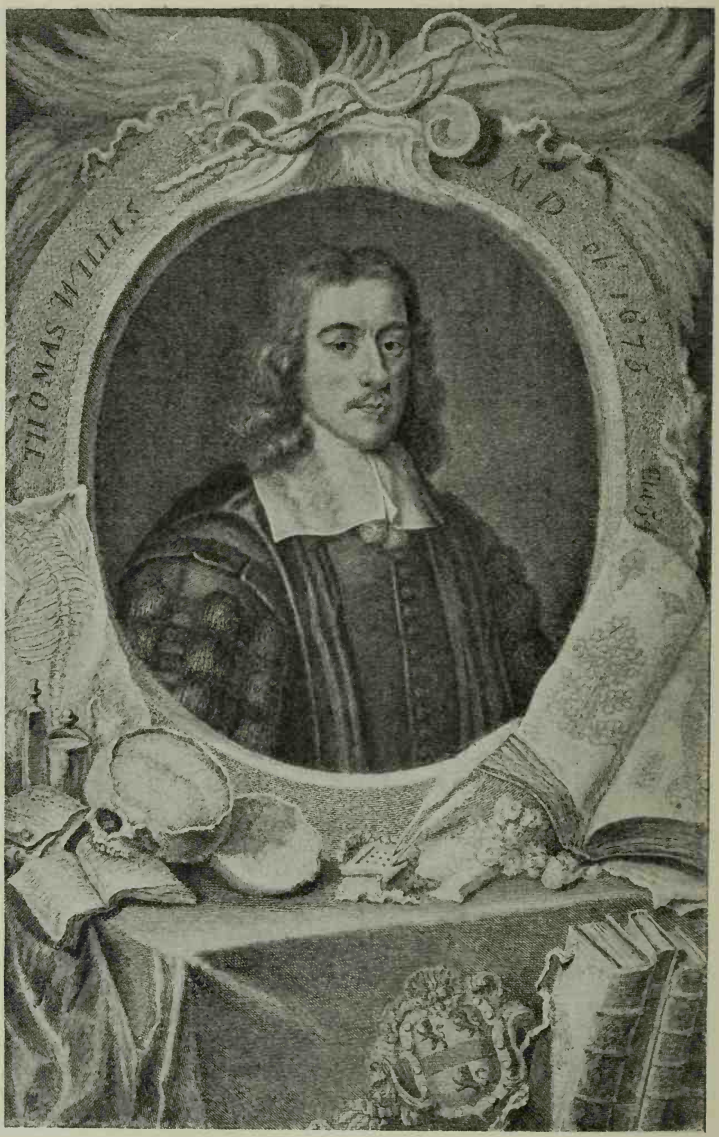


FIG. 1. THOMAS WILLIS.

SURGICAL ASPECTS OF TYPHOID AND PARATYPHOID FEVERS

CHAPTER I

HISTORICAL SKETCH

An inquiry into the early records of Typhoid Fever in this country opens some interesting pages of English history. In war, we first find evidence of the disease in 1643, when, during the Civil War, the ranks of the Parliamentary forces were thinned by an outbreak of disease, from which King Charles's followers also suffered, and which they carried with them to the Royalist stronghold at Oxford. Here Thomas Willis was serving the King in the University Legion. He was then a student of medicine, and from the detailed account which he gave of this epidemic in his classical work *De Febribus* there is no doubt that it was typhoid fever.

This is the same Willis whose name is so familiar to us from the anastomosis of the main cerebral arteries at the base of the brain being called after him. He became a great man, was a contemporary of the great Sydenham ; and in addition to his work on fevers, being one of the first to describe typhoid fever in epidemic form, he made valuable contributions to Anatomy and Physiology, and was the first to recognise diabetes mellitus.

FIG. 2. HENRY, PRINCE OF WALES.

Since the time of this, its first known appearance in war, typhoid fever has been a menace to armies in all campaigns of any duration, and has often cost the British Army dear.

In peace as well as in war, however, we have had to suffer as a nation from the ravages of this disease, which has attacked members of our Royal House on more than one occasion. The very first case on record in England which allows of clear demonstration was that of Henry, Prince of Wales, eldest son of King James I, who died in 1612 of a fever epidemic at that time. Prince Henry was a young man of considerably greater ability than his brother Charles, and had he not died when only eighteen years of age it might have been that the acute differences which led to Civil War would have been settled in another way. This is not at all certain, however, since historians tell us that Henry was just as headstrong as Charles, and held equally strong views with regard to the Royal Prerogative.

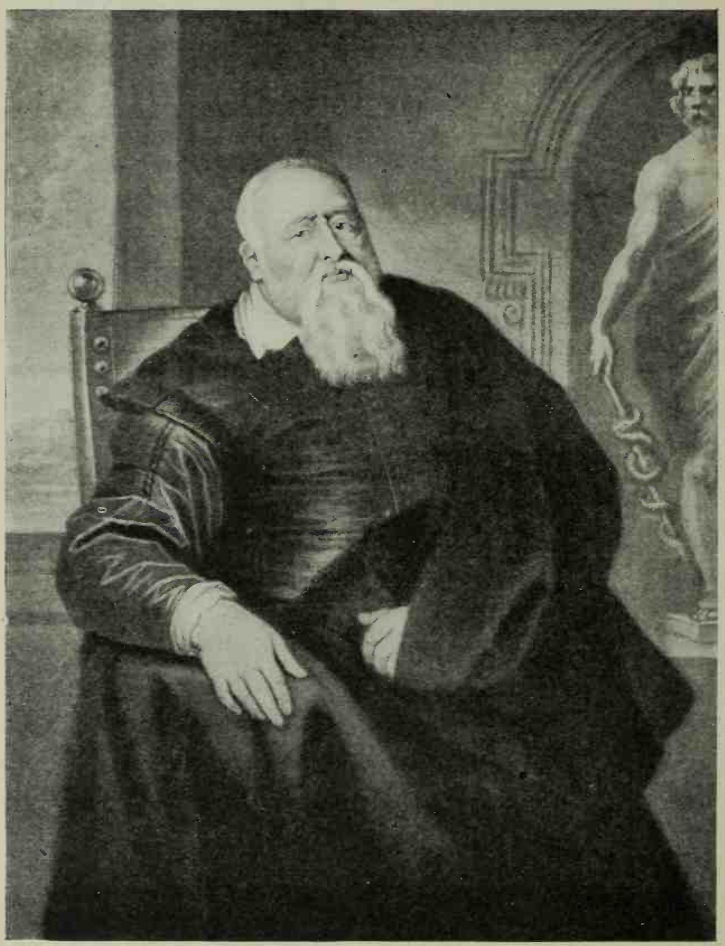


FIG. 3. SIR THEODORE DE MAYERNE.

For the careful account of Prince Henry's illness and the description of the appearances found at the post-mortem examination, which were published mainly on account of the difficulty of diagnosis and of prevailing suspicions that the Prince had died of poisoning, we are indebted to that distinguished French physician, Sir Theodore de Mayerne, who was in attendance on the Prince during the last part of his illness. The exact account left by Sir Theodore de Mayerne has enabled Dr. Norman Moore to recognise the illness as being due to typhoid fever, although the physicians of the time could not go further than to say that it was a fever. ("On the Illness and Death of Henry, Prince of Wales: An Historical Case of Typhoid Fever." Norman Moore, *Saint Bartholomew's Hospital Reports*, vol. xvii. 1881.)

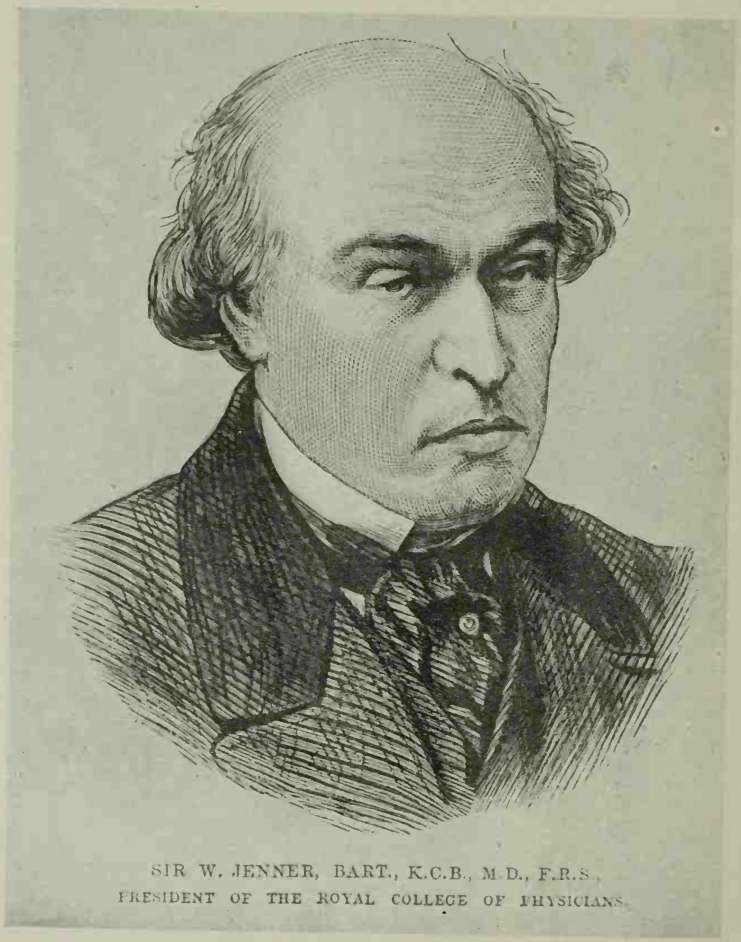


FIG. 4. SIR WILLIAM JENNER.

If we leave this period and pass to a time within living memory, we find typhoid fever causing the Nation to mourn the loss of Prince Albert in 1861, while the late King Edward was brought to the brink of the grave by the same disease ten years later. Among the physicians in attendance on these Royal patients was Sir William Jenner, whose work at the London Fever Hospital from 1847 to 1851 did so much to help in the differentiation of the continued fevers, and, to use his own words, "went to prove incontestably, so far as induction can prove the point, that the specific causes of typhus and typhoid fevers are absolutely different from each other," a question which was still a source of dispute in England in spite of the work of Louis in Paris, Gerhard and Stillé in America, and the researches of Alexander Stewart in Glasgow and London.

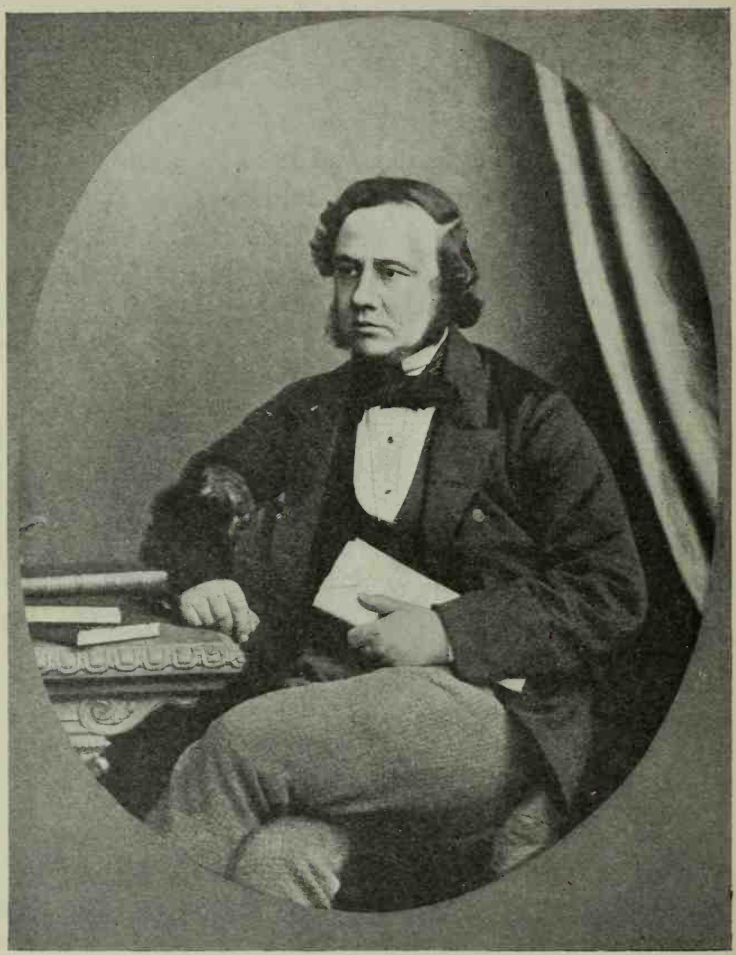


FIG. 5. DR. CHARLES MURCHISON.

Later Dr. Charles Murchison, when he was on the staff of the Middlesex Hospital, and, like William Jenner on that of the London Fever Hospital, made valuable contributions to the study of the continued fevers. Murchison's classical work on *The Continued Fevers of Great Britain*, published in 1862, became

Fig. 6. Dr. William Cayley.

at once a standard authority, and still maintains a prominent place in the literature of the subject. The third edition of this work was edited, after Murchison's death, by another physician to the Middlesex and London Fever Hospitals, a great authority on Infectious Fevers who died only in 1916, Dr. William Cayley.

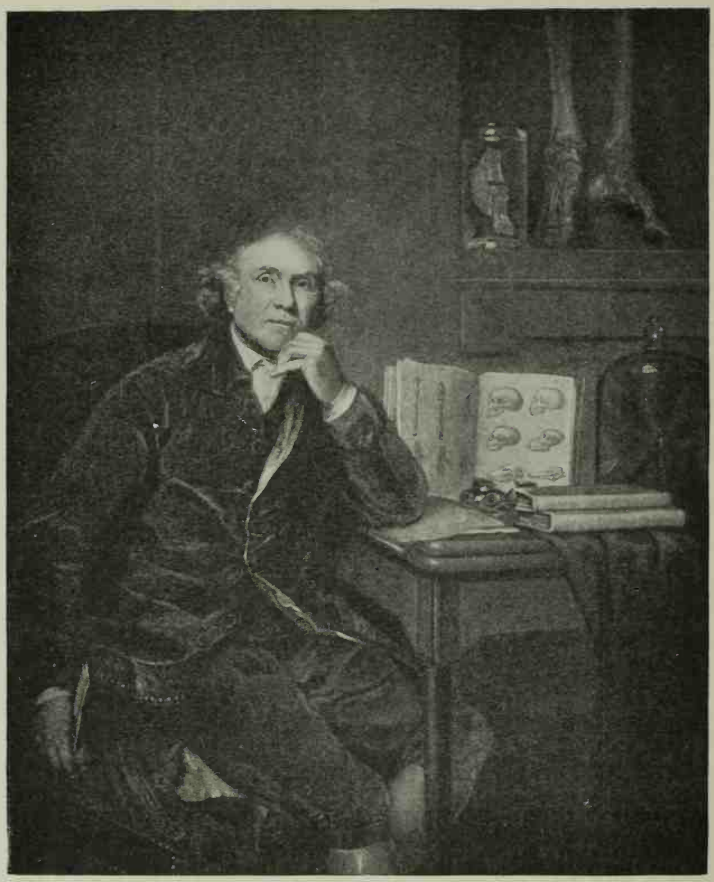


FIG. 7. JOHN HUNTER.

By the time of the Prince Consort's illness, a clear distinction had been established between typhus and typhoid fevers, and the clinical characteristics of the continued fevers generally were also well recognised. This position was attained mainly through careful clinical research and the methodical investigation of morbid anatomy, to which work it would be surprising indeed if we did not find some contribution from John Hunter.

John Hunter must have met with typhoid fever when serving with the army during the Seven Years' War, for then, as now, war drew men to work in channels to which they were not accustomed in times of peace. In 1762, in a letter from Portugal to his brother William, John Hunter writes, "I am now applying for Phisician, if I get that I shall be a Dr. as well as the best of you." He became indeed, as Stephen Paget says, "Anatomist, biologist, naturalist, physician, surgeon, and pathologist, all at once, and all in the highest." And as Osler said (when writing of "The Influence of Louis on American Medicine," *Johns Hopkins Hospital Bulletin*, 1897), "John Hunter, the greatest anatomist and medical thinker of modern times, to whose broad vision disease was but one of the processes of nature to be studied, was as a voice crying in the wilderness to the speculative, theoretical physicians of his day."

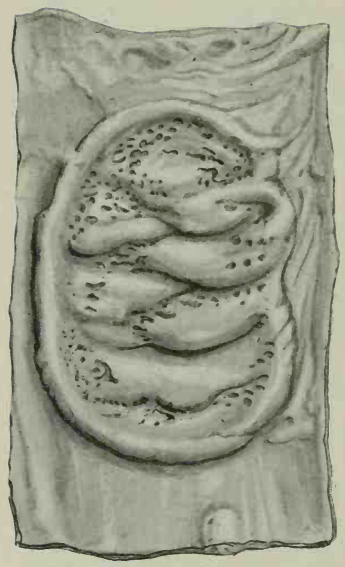


FIG. 8. Portion of ileum showing great enlargement of a Peyer's patch. (From Hunter's original collection. Museum of the Royal College of Surgeons of England. Specimen No. 2508.)

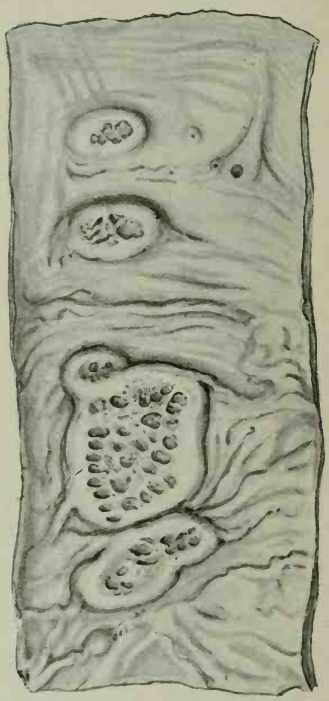


FIG. 9. Portion of ileum showing enlargement of several small Peyer's patches. (From Hunter's original collection. Museum of the Royal College of Surgeons of England. Specimen No. 2509.)

Reference to the Hunterian manuscripts shows that Hunter, in the course of his "Dissections of Morbid Bodies," gave close attention to the morbid anatomy of this disease, and he also gave clinical descriptions of cases of fever with ulceration of the intestine which we now know to have been typhoid. In the museum of the Royal College of Surgeons are fifteen specimens of the intestinal lesions from Hunter's original collection. The specimens show the various stages of enlargement and ulceration of Peyer's patches and of the solitary follicles with the formation and separation of sloughs and the process of healing. The accompanying drawings are of some of the most interesting of Hunter's specimens.

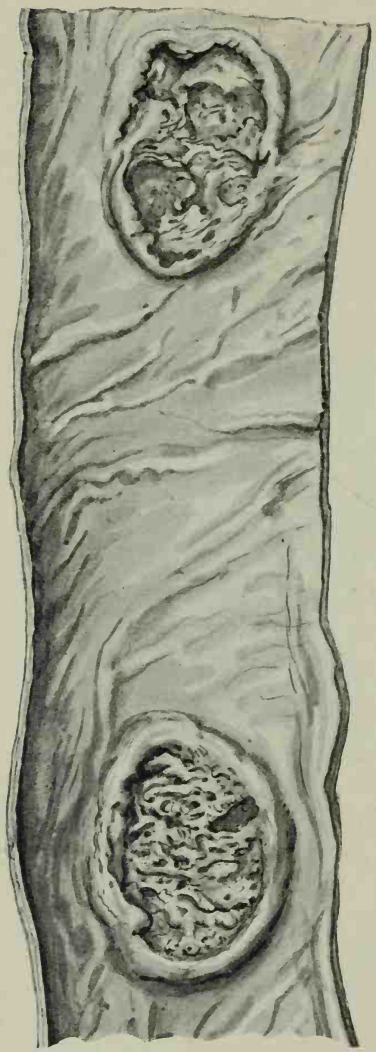


FIG. 10. Portion of ileum exhibiting an advanced stage of typhoid ulceration. (From Hunter's original collection. Museum of the Royal College of Surgeons of England. Specimen No. 2497.)

FIG. 11. Cæcum, last part of ileum and appendix in which are several small deep typhoid ulcers nearly healed. (From Hunter's original collection. Museum of the Royal College of Surgeons of England. Specimen No. 2504.)

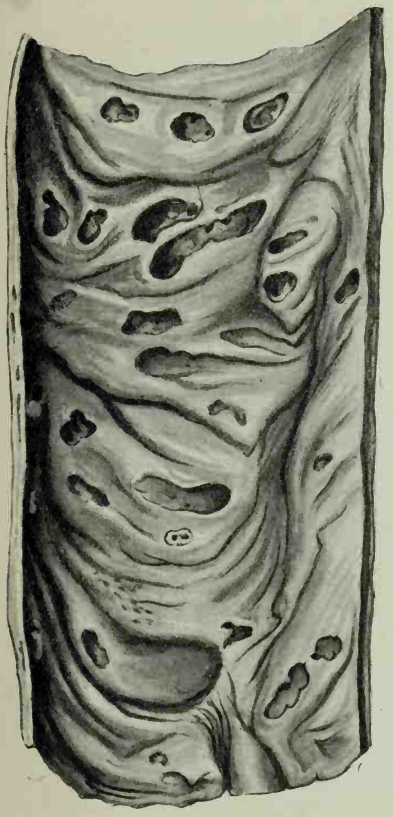


FIG. 12. Portion of colon showing ulcers partly healed. This specimen may have been from a case of paratyphoid fever, and may be compared with Fig. 20. (From Hunter's original collection. Museum of the Royal College of Surgeons of England. Specimen No. 2502A. The drawing is copied from the original by Wm. Clift in Matthew Baillie's *Engravings of Morbid Anatomy.*)

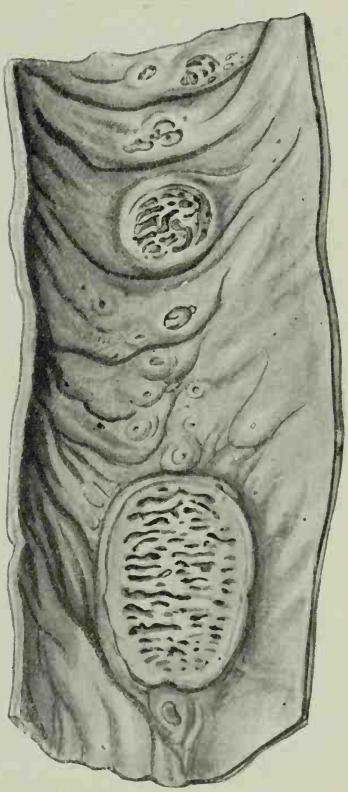


FIG. 13. Portion of ileum showing two Peyer's patches with hyperplasia and early necrosis. (From Hunter's original collection. Museum of the Royal College of Surgeons of England. Specimen No. 2514. The drawing is copied from the original by Wm. Clift in Matthew Baillie's *Engravings of Morbid Anatomy.*)

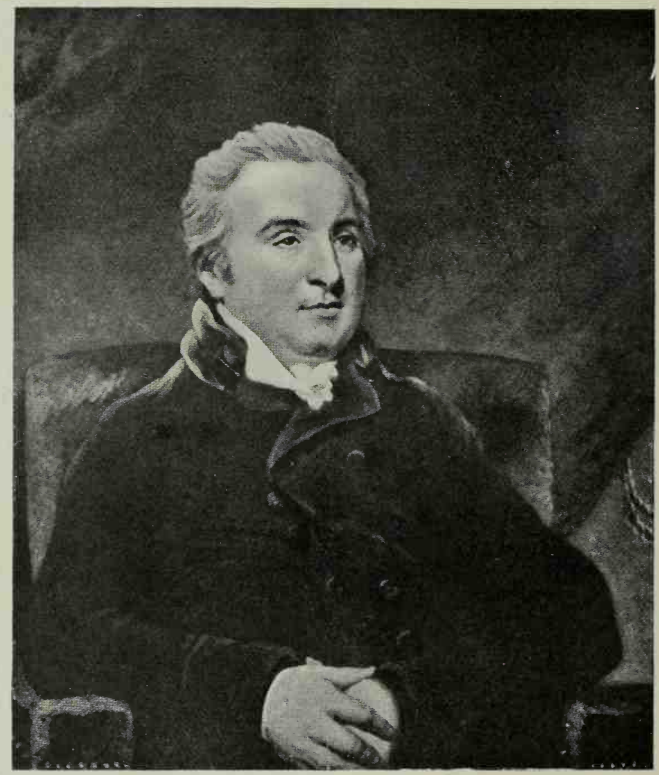


FIG. 14. MATTHEW BAILLIE.

The last two drawings (Figs. 12 and 13) are of special interest, for they are copied from the *Engravings of Morbid Anatomy*, published in 1803 by Hunter's nephew, Matthew Baillie, who was the first medical writer to treat morbid anatomy as a subject by itself, having published his *Morbid Anatomy of Diseases* in 1795. Baillie gives exact descriptions of the specimens, and his descriptions are still valuable, though he wrote them without the necessary pathological knowledge of the diseases which had caused the lesions.

Fig. 12, with ulcers in the colon, might be thought to represent dysenteric rather than typhoid ulceration. Curiously enough the patient was said to have had dysentery, but Paget and Goodhart classed the specimen as a typhoid one, and comparing it with some of those from the present series it might well have been from a case of paratyphoid fever. (See Fig. 20.)

FIG. 15. WILLIAM CLIFT.

These two illustrations (Figs. 12 and 13) have another great point of interest, as the drawings for the original engravings from which these are copied were by William Clift, Hunter's devoted servant, and the faithful guardian of the Hunterian Collection from the time of Hunter's death in 1793 until the Royal College of Surgeons came into existence in 1800, when he became the first Conservator of the Museum.

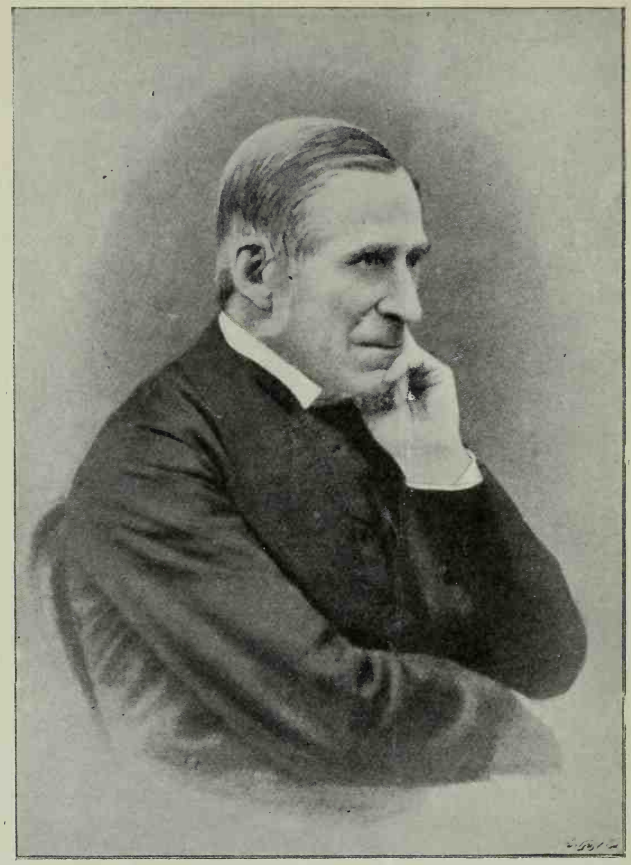


FIG. 16. SIR JAMES PAGET.

After Hunter, Paget. To him we owe so much for his work in the museum of the Royal College of Surgeons and for the careful classification and description of pathological specimens in the light of knowledge not available in Hunter's time. Sir James Paget was amongst the first to realise the large and interesting field for surgical investigation in the complications and sequelæ of the continued fevers. In his *Clinical Lectures and Essays*, published in 1875, he writes, "The sequelæ of scarlet fever are commonly enumerated; those of typhoid fever, especially those seen in surgical practice, are scarcely less numerous, but seem less known."

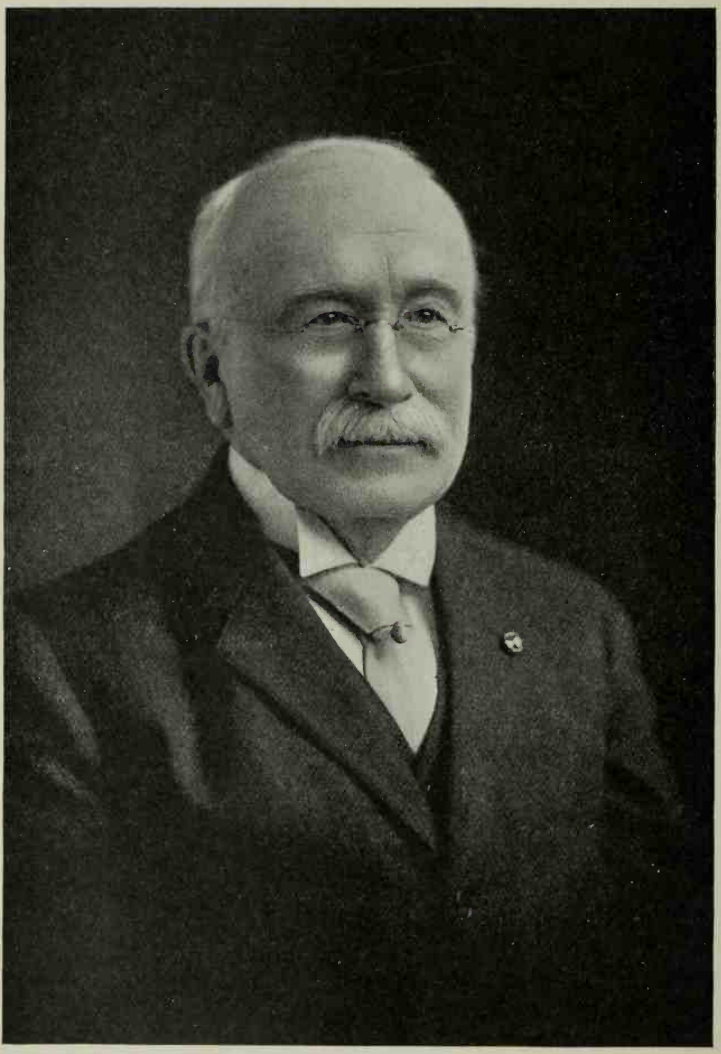


FIG. 17. PROFESSOR W. W. KEEN.

The first surgeon to give this subject the careful study which Paget saw it deserved was W. W. Keen of Philadelphia, who in 1876 delivered the Toner Lecture on "The Surgical Complications and Sequels of the Continued Fevers," and in 1898 published his well-known book on *The Surgical Complications and Sequels of Typhoid Fever.*

Between these two dates an epoch-marking discovery was made, for in 1880 Eberth demonstrated the typhoid bacillus in the spleen and intestinal ulcers, while in 1884 Gaffky gave a detailed account of this bacillus, and in 1885 it was isolated from the blood by Fraenkel and Simmonds. With the discovery of other closely allied organisms, such as the bacillus coli communis (by Escherich in 1885), the bacilli of meat poisoning (the bacillus suipestifer in 1885 and the bacillus enteritidis of Gärtner in 1888), and others, it became increasingly evident that there existed a group of bacilli which were practically identical morphologically, but which differed in their pathogenetic properties. A new chapter in the history of the typhoid fever problem was opened, however, by the discovery of organisms which not only closely resembled the bacillus typhosus, and yet were distinguishable from it, but which also produced identical symptoms and post-mortem appearances.

In 1896 Achard and Bensaude isolated organisms which they called paratyphoid bacilli from two patients who had been diagnosed as suffering from typhoid fever—in one case the bacilli were obtained from the urine, and in the other from a suppurative arthritis of the sterno-clavicular joint. In 1898 Gwyn cultivated a similar bacillus from the blood of a patient with symptoms of typhoid fever, and in 1901 Schottmüller described five cases of what was apparently typhoid fever, in which he had found two species of paratyphoid bacilli. These organisms were named by Brion and Kayser the bacillus paratyphosus A and the bacillus paratyphosus B, forming with the bacillus typhosus a typhoid sub-group of the large coli-typhoid group of organisms. By painstaking research these organisms have been carefully classified, and in 1912 Bainbridge in his "Milroy Lectures" (*The Lancet*, March 16th, 23rd, and 30th, 1912) demonstrated that the paratyphoid bacilli were distinct from the food-poisoning bacilli with which they had been confused by German writers. This classification represents the present state of our knowledge of the typhoid group of organisms, but it has still to stand the test of further bacteriological and clinical research.

CHAPTER II

INTRODUCTORY

It has then been established that the bacilli of the typhoid sub-group are distinct specific micro-organisms, and the diseases to which they give rise are known as typhoid fever, paratyphoid A and paratyphoid B.

The symptoms of the three diseases are the same; individual cases are indistinguishable clinically, and their post-mortem appearances are identical. The following plates represent the intestinal lesions of cases of this series. I am indebted to Major-General Sir Bertrand Dawson and Captain T. H. Whittington for permission to publish them, as well as Fig. 23 and Plate I.

When numbers of cases are analysed, it is seen that paratyphoid fever is a much milder disease than typhoid. Fever due to the bacillus paratyphosus A is the prevailing type of the disease in the East, and has been studied in India especially by officers of the Royal Army Medical Corps (Grattan and Wood, Firth, Harvey, and others), and in Ceylon by Castellani, whose work has thrown so much light on the problems of the typhoid group of fevers. Paratyphoid A is a milder disease than paratyphoid B, which is the form of paratyphoid

FIG. 18. *Paratyphoid B:* Lower portion of ileum showing numerous ulcers, from which the sloughs have completely separated.

FIG. 19. *Paratyphoid B :* A Peyer's patch in the ileum, showing hyperæmia and early necrosis.

(From *The Quarterly Journal of Medicine.*)

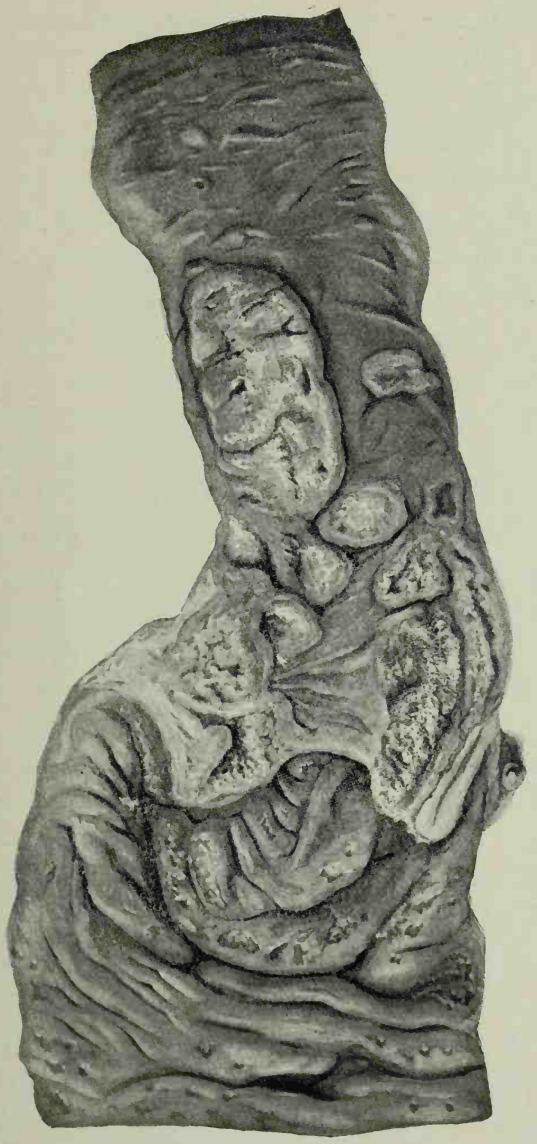


FIG. 20. *Paratyphoid B:* Ileum and cæcum. The ulcers in the ileum are deep and have separated in part. The cæcum is riddled by small ulcers, some of which have coalesced.

(From *The Quarterly Journal of Medicine.*)

fever usually encountered in the Western hemisphere. The clinical picture of paratyphoid fever met with amongst the British troops in France has been described by Sir Bertrand Dawson (*Proceedings of the Royal Society of Medicine*, 1915, vol. ix., Medical Section, pp. 1–9) and by Torrens and Whittington (*British Medical Journal*, November 13th, 1915), while the bacteriological problems have been dealt with from time to time by Professor Dreyer and others.

In the words of Sir Bertrand Dawson: "Looked at broadly, paratyphoid is typhoid fever in miniature. A patient invaded by it stands to be ill less severely and for a shorter time, is less prone to complications, and, *a priori*, less likely to die. It presents a clinical picture which is suggestive, though not absolutely distinctive, and its certain diagnosis from typhoid can only be made with the aid of bacteriological findings"; but again he gives warning that, "Though habitually milder, paratyphoid may present the gravity of severe typhoid." It is of some importance, therefore, in order to determine the danger of an outbreak of disease due to typhoid group infection, or to estimate the prognosis in an individual case, that the infecting organism should be ascertained.

For my analysis of the surgical complications of these fevers I have taken the cases admitted to the largest British Isolation Hospital in France during the first two years of its existence. I have not included 184 non-fatal cases which were not

diagnosed more definitely than as "Enteric Group," leaving 2,500 diagnosed as either Typhoid, Paratyphoid A, or Paratyphoid B. (See Table I.) During the same period 1,274 of the cases admitted as "Suspected Enteric" were found not to be Enteric Group cases. In many cases it has not been possible to isolate the infecting bacillus from the blood, for the bacillæmia in these fevers, especially in inoculated individuals, is only transient, being present for the shortest time in the case of paratyphoid A. Very often the patients were admitted to the isolation hospital some days after the onset of the disease, and the bacteriologists did not succeed in obtaining the bacilli from blood, stools, or urine, especially in the case of those protected by the triple "T.A.B." vaccine. In these cases we have been dependent for the confirmation and establishment of the diagnosis on examination of the patient's serum and the determination of its agglutinating power on the organisms of the typhoid group.

TABLE I

Disease.	Number of Cases.	Deaths.	Mortality per cent.
Typhoid—inoculated	821	27	3·28
Typhoid—not inoculated	297	57	19·19
	1118	84	7·51
Paratyphoid A—inoculated	123	0	0·00
Paratyphoid A—not inoculated	221	1	·45
	344	1	0·29
Paratyphoid B—inoculated	239	1*	0·41
Paratyphoid B—not inoculated	799	17	2·12
	1038	18	1·68
Totals	2500	103	4·12

* This patient had lymphosarcoma.

Serum Diagnosis.—The application of the agglutination test to the diagnosis of typhoid fever by Widal in 1895 made it possible to confirm a diagnosis without isolating the infecting organisms from the blood. This reaction enables bacteriologists to test a patient's serum with various bacilli, and from the agglutinating effects of the serum to state which bacillus is the infecting agent. Moreover, it also enables them to identify bacilli by ascertaining how they are affected by sera which are known to contain certain specific agglutinins. This is merely a broad statement of the case. The actual investigation of a patient's serum is beset with many difficulties. Infection with one organism may give rise to the production of agglutinins of other allied organisms as well as its own specific agglutinins, though not to the same extent. Then again, agglutinins are produced in response to protective inoculation, a fact which makes it more difficult to determine whether an inoculated individual is suffering from a typhoid group infection, and, if so, to which organism the infection is due. The difficulties of diagnosis, however, have been overcome. By means of Professor Dreyer's delicate technique the amount of any specific agglutinins present in a patient's serum can be determined with accuracy; and, with few exceptions, in the case of inoculated individuals, by the repeated measurement and the study of the variations in the agglutinating power of a patient's serum it can be shown what organisms of the typhoid group, if any, are the cause of his

illness. An authoritative statement of the value of the agglutination method of diagnosis in inoculated individuals has been published recently by Captain H. Marrian Perry, R.A.M.C. (*The Lancet*, April 27th, 1918).

Blood Picture.—Before discussing the surgical complications a word about the blood picture associated with these infections. As in tuberculosis and influenza and other diseases caused by bacilli, the reaction of the cellular elements of the blood to infection by typhoid or paratyphoid bacilli consists of an increase in the number of lymphocytes at the expense of the polymorphonuclear cells. This change is progressive and, owing to the diminution in the number of polymorphonuclear cells an actual leucopenia may be produced. When surgical complications supervene, this leucopenia is replaced by a leucocytosis with an increase in the relative and absolute number of the polymorphonuclear leucocytes, but the number of these cells and the degree of leucocytosis are not so high as those usually met with in suppurative conditions not associated with a bacillary infection.

CHAPTER III

GENERAL REVIEW OF SURGICAL COMPLICATIONS

Reference to the literature and an examination of Table II, which shows the surgical complications met with in the present series, make it clear that no part of the body is safe from the effects of a typhoid or paratyphoid infection. As Murchison said, "There is no disease which exhibits a more protean character, from predominance of certain symptoms, and from the presence of complications." We now know that this is to be explained by the fact that the disease is essentially a septicæmia. The portal of entry is some part of the alimentary tract, for the disease is known to be conveyed by food and water. The bacilli are found in the blood in over 90 per cent. of cases of typhoid amongst non-inoculated patients, and have been shown to be present in the blood during the incubation period, but it is not until the first and second weeks or later that the bacilli appear in the fæces and urine. All parts of the body are invaded therefore by the bacilli, and in some organs and tissues a special defence is put up and the signs of battle result. Inflammatory reactions of a defensive character occur, and the typical lesions of the

disease are produced. The most striking of these are changes of a protective character in the blood, inflammation of the lymphoid tissue of the intestine (especially of the lower part of the ileum) and mesenteric glands, great enlargement of the spleen to deal with the bacterial invasion and filter off the organisms from the general circulation, and parenchymatous changes in other organs and tissues, especially the liver. Many parts of the body are only rarely the seat of an inflammatory process, and these inflammatory conditions are generally described as complications.

In some cases the characteristic symptoms or lesions are absent, but evidence of a typhoid group infection is found in some suppurative or other inflammatory lesion. More particularly are these found in the liver and bile passages in which cases presumably the spleen has separated the infecting organisms from the blood stream, and they have passed to the liver and bile passages where the infection has become localised. In this way it is that individuals may become "carriers" of the bacilli without ever having had typhoid fever in a form to be recognised, though they have probably suffered from it in its mildest manifestations.

In some of the complications the specific organisms of the typhoid group are found either in pure culture or with pyogenic cocci, while in others typhoid group bacilli are not found, but other organisms, such as the ordinary pyogenic cocci or the bacillus coli communis, are isolated. The bacteriology of

the pus and other inflammatory products obtained from complications of the typhoid group of fevers depends to a large extent on their time of onset. If they occur late in the disease or in convalescence, or after recovery, the specific organisms are usually found in pure culture. If, however, complications arise during the height of the fever, when numerous portals of entry, such as the intestinal ulcers, bedsores, boils, etc., are open for the admission of any organisms which are ready to take advantage of a lowered resistance, it is usual for the pus and other inflammatory products obtained to yield only pyogenic cocci, generally staphylococci. Any organisms of the typhoid group which may have been present originally in the part, may have been outgrown by the pyogenic cocci. The bacteriology depends also to some extent on the part affected—for example, it is usual to find the specific bacteria of the disease in such lesions as abscesses of the spleen and liver, and in other abdominal abscesses at whatever period of the disease they may develop.

I have included in Table II some conditions such as tonsillitis and intestinal hæmorrhage, which are not directly surgical, but, since they bear a distinct etiological relationship to complications which are purely surgical in their nature and treatment, I think it well to include them for purposes of comparison. A point which is obvious from the table is that typhoid is by far the most serious of these infections, and that surgical complications as

Disease.	Typhoid Inoculated.	Typhoid Not Inoculated.	Paratyphc Inoculat
Cases	821	297	123
Deaths	27	57	0
Mortality	3·28%	19·19%	0
Hæmorrhage	10 = 1·21%	40 = 13·46%	0
Perforation	3 = ·36%	6 = 2·02%	0
Appendicitis	4 = ·48%	2 = ·67%	0
Peritonitis without perforation	0	0	0
Splenic Abscess	1 = ·12%	0	0
Liver Abscess	0	0	0
Suppurating Mesenteric Glands	1 = ·12%	1 = ·33%	0
Cholecystitis	4 = ·48%	2 = ·67%	0
Gallstones	1 = ·12%	0	0
Stomatitis	0	0	0
Noma	0	1 = ·33%	0
Tonsillitis	10 = 1·21%	4 = 1·34%	1 = ·8
Quinsy	1 = ·12%	0	0
Deafness	1 = ·12%	1 = ·33%	0
Otitis Media	3 = ·36%	3 = 1·01%	0
Parotitis	0	6 = 2·02%	0
Laryngitis	2 = ·24%	8 = 2·69%	0
Lung Abscess	0	0	0
Empyema	0	0	0
Venous Thrombosis	7 = ·85%	10 = 3·36%	0
Urinary Complications	1 = ·12%	4 = 1·34%	0
Epididymo-Orchitis	0	0	0
Bone Lesions	1 = ·12%	3 = 1·01%	0
Arthritis	2 = ·24%	0	0
Muscle Lesions	0	3 = 1·01%	0
Other Abscesses	7 = ·85%	5 = 1·68%	0
Boils	3 = ·36%	2 = ·67%	0
Erysipelas	0	5 = 1·68%	0
Totals	62 = 7·55%	106 = 35·69%	1 = ·8

* This patient had lympl

a whole are commoner in typhoid than in either paratyphoid A or paratyphoid B. This is equally true whether the complications are due directly to the specific infection or are only caused indirectly by it by lowering resistance and providing portals of entry for other micro-organisms. From the general character of the complications met with in this series and from published accounts of paratyphoid cases, it may be said with certainty that any complication that has been met with in typhoid fever may also be met with in paratyphoid fever. In view of the marked difference in the mortality of these diseases and the incidence of complications in the different fevers of the typhoid group, it is important for a definite diagnosis to be made. Statistics referring to series of cases of typhoid group infection in which the cases have not been differentiated into typhoid or paratyphoid A or B are only of minor value compared with those in which this distinction has been made.

The cases of paratyphoid A are comparatively few in this series, but it is clear that the disease, as met with amongst the British troops in France, is a long way the mildest of the three. Considering the mild character of the disease, and the small incidence of intestinal hæmorrhage, which may be taken as an indication of the severity of the intestinal lesions, the fact that the percentage of perforations in paratyphoid A was greater than that in paratyphoid B is probably accidental. When dealing with comparatively small numbers, one case with

complications makes a big difference in the percentage.

In paratyphoid B one of the striking features is the extent to which the large intestine is involved, some specimens almost resembling the condition of the bowel met with in cases of dysentery—it may be, as suggested on page 35, that Hunter's specimen shown in Fig. 12 was from a case of paratyphoid fever.

Paratyphoid A and B gave rise to cholecystitis as often as did true typhoid, and paratyphoid B proved the most irritating to the urinary tract. Torrens and Whittington (*British Medical Journal*, Nov. 13th, 1915) called attention to the tendency to suppuration in paratyphoid B, and this is borne out by the number of suppurative complications. The pyogenic properties of the bacilli of the typhoid group as a whole have been fully demonstrated, and in this series at any rate the bacillus paratyphosus B led to suppuration more often than the bacilli of typhoid or paratyphoid A.

Protective Inoculation.—This is not the place to speak of the small incidence of this group of fevers amongst the British troops in France, but we must pay a tribute to the efficiency of the sanitary service as well as to the effects of inoculation in this direction. With regard to the effects of inoculation on the course of the disease, by far the most striking point in this review is the remarkable difference in severity of illness, incidence of complications and mortality between the inoculated and

the non-inoculated. In compiling the statistical tables I have given the widest possible interpretation to the term "inoculated," and have included as inoculated all who have had any specific vaccine at all, whether they have had a full dose or not, and also whether or not it has been administered within the time for which it is claimed that protection is conferred. It would have made the tables too elaborate to have done otherwise. Patients with paratyphoid fever who have only been inoculated against typhoid are counted as non-inoculated. The statistical tables are convincing enough, but the difference in severity of illness is considerably more striking when you are brought into actual contact with the patients, for most of the inoculated are obviously suffering from a relatively mild disease. The mortality was five times as great in the unprotected as in the protected. This result may well lead to some reflection especially when it is remembered that the hospital was situated in Boulogne Base. There, under the eyes of the originator of anti-typhoid inoculation, Sir Almroth Wright, we have seen some of the benefits of protective inoculation against these fevers; we have seen them in the country of the great Pasteur and within a stone's throw of France's memorial to Edward Jenner, the founder of scientific protective vaccination.

The pedestal of the statue to Jenner bears inscriptions which are of such great interest that one is constrained to reproduce them.

FIG. 21.—Statue of Jenner in Boulogne.

On the back of the pedestal is carved :—

A

EDWARD JENNER

LA FRANCE RECONNAISSANTE

On the front of the pedestal is the following:

"Ce monument a été élevé de concert par la Ville de Boulogne-sur-Mer et la Société des Sciences Industrielles, Arts et Belles Lettres de Paris en l'honneur de Edward Jenner, auteur de la découverte de la vaccine."

And one side of the pedestal is inscribed as follows :

"William Woodville, Médecin de l'Hôpital des Varioleux de Londres, apporta au peuple Français, malgré l'état de guerre, la découverte de Jenner et pratiqua les premières inoculations à Boulogne-sur-Mer le 27 Prairial, An VIII (19 Juin 1800). Le vaccin recueilli par le Dr. Nowel, fut envoyé à Paris, où Woodville l'inocula de nouveau en Thermidor suivant."

In these times of war Anstey's ode to Jenner becomes again particularly apt. Ring's translation and paraphrase runs as follows :—

"While deathless heroes, who maintain our fame,
And add new glories to the British name,
Around their brows unfading laurels twine,
The Civic Crown, O Jenner, shall be thine."

With regard to inoculation there is another point, which is possibly one of the most important. The specific bacteria are not found in either blood, fæces, or urine with anything like the same frequency, nor in such numbers in the inoculated as in the non-inoculated, since the microbes are killed more rapidly and effectually owing to protective

substances being present in the body ready for the attack. It follows that the sequels and after effects of typhoid and paratyphoid infection are fewer in the inoculated, and there is less chance of infected individuals becoming "carriers" of the bacilli. Therefore not only is inoculation of benefit to the individual and to the army in largely preventing infection, as shown by the small incidence of cases, in controlling the disease in those who become infected and making the attack milder and complications and sequels less frequent, and also considerably reducing the mortality, but in addition it may be of benefit to the community at home by diminishing the number of men who become "carriers."

CHAPTER IV

THE ALIMENTARY TRACT

The Œsophagus.—Ulceration of the œsophagus has been noted in rare instances at post-mortem examination of cases of typhoid fever. The ulcers may be due to infection with typhoid group bacilli or pyogenic cocci. During the course of the disease some pain or difficulty in swallowing may be observed, and this may be due to glossitis, tonsillitis, pharyngitis, or to ulceration of the œsophagus. Œsophageal stricture has been recorded from time to time as a sequel of typhoid fever. Some cases have yielded to dilatation, and in others gastrostomy has been necessary.

The Stomach.—In a few cases of typhoid fever, ulceration of the stomach has been found at autopsy. Hæmatemesis has been described in rare instances, and perforation of a typhoid ulcer of the stomach has occurred. There were no instances of gastric complications in the present series, though vomiting was noted in some cases. As a rule vomiting should be regarded as a sign of some intestinal complication, unless it can be accounted for by some increase in the quantity or change in the character of the patient's diet.

Intestinal Hæmorrhage.—Taking the average of a large number of collected statistics, intestinal hæmorrhage is found to occur in about 7 per cent. of cases of typhoid fever. In the 297 non-inoculated typhoid cases of this series hæmorrhage occurred in forty, or 13·46 per cent., an unusually high proportion. There were, however, only sixty-seven cases in the whole series of 2,500 cases of enteric group infection, which works out at only 2·68 per cent. Hæmorrhage occurs most commonly in the second and third weeks of the disease; it is the most frequent complication of these fevers, and a large proportion of the cases are fatal; of the sixty-seven patients who suffered from intestinal hæmorrhage, death occurred in thirty, or 44·8 per cent. Intestinal hæmorrhage is directly and immediately fatal in only very few cases, but is a symptom of grave omen, for it occurs as a rule in the most severe cases, and often seriously affects the condition of the patient. It was noted as a complication in nearly 30 per cent. of the total fatal cases in the present review.

It has been suggested that hæmorrhage from a typhoid ulcer might be dealt with by operation, but this course can seldom be practicable, and circumstances would have to be very unique and favourable to warrant such a procedure. The main surgical interest of intestinal hæmorrhage in typhoid and paratyphoid fever is that it renders the patient more liable to other complications which are essentially surgical in their nature and treatment.

It is important also to be specially on the look-out for symptoms of perforation of the intestine in patients who are suffering or have suffered from intestinal hæmorrhage, a point which will be discussed when dealing with intestinal perforation.

A patient suffering from intestinal hæmorrhage should be kept absolutely quiet. The coagulability of the blood should be increased by the administration of calcium salts, and for the anæmia and collapse following a serious hæmorrhage transfusion of blood may be necessary. Opium should not be given, for it may mask important symptoms, since it is not uncommon for perforation to follow or to occur at the same time as intestinal hæmorrhage.

Perforation of the Intestine.—Perforation of the intestine is at once the most formidable and most urgent of the surgical complications of typhoid and paratyphoid fever, for not only is the patient's life immediately endangered, but it is essential to his recovery that the lesion be recognised and dealt with promptly.

Incidence.—The usual frequency of intestinal perforation in typhoid fever is about once in every thirty to forty cases, but it varies in different years and different epidemics. It is rare in children, and commoner in men than in women. It will be seen, therefore, from a study of Table III, that in the present series the incidence has been very small, especially when it is borne in mind that only 14 of the 2,500 cases were women, and the great majority of the patients were between twenty and

thirty years of age, during which decade perforation of the intestine occurs most frequently.

TABLE III

Disease.	No. of Cases.	Cases of Perforation.	Percentage.	Operated on.	Recovered.	Died.	Day of disease on which perforation occurred.
Typhoid inoculated .	821	3	0·36	2	—	3	13th, 17th, 22nd, 27th, 28th, 31st, 39th, 51st, 70th.
Typhoid not inoculated . .	297	6	2·02	1	—	6	
Paratyphoid A inoculated . .	123	—	—	—	—	—	25th, 30th.
Paratyphoid A not inoculated .	221	2	0·90	1	1	1	
Paratyphoid B inoculated . .	239	—	—	—	—	—	12th, 23rd, 33rd.
Paratyphoid B not inoculated .	799	3	0·37	2	—	3	
Total . . .	2,500	14	0·56	6	1	13	

In view of the incidence of other complications in paratyphoid A the high percentage of perforation is probably accidental, and therefore deceptive. A larger number of cases would have to be analysed to obtain a true idea of the frequency of intestinal perforation in this disease.

Amongst the few fatal cases of typhoid in which no post-mortem examination was made, there were two in which it is possible that perforation occurred as a terminal event, and these are not included in the table, as the diagnosis was not confirmed.

Also in one patient who recovered from typhoid, some of the hospital staff thought that perforation had occurred. The possibility of recovery from perforation without operation, though of very exceptional occurrence, must be admitted.

Time of Onset.—In typhoid fever, as a rule, most perforations occur in the third week of the disease. In ambulatory cases it may be the first sign. It has been reported as early as the 8th, and as late as the 110th day. In the present series (Table III) it will be seen that eight out of the fourteen cases occurred during the 4th and 5th weeks, while one was as early as the 12th day and another as late as the 70th day.

There is fresh inflammation of the lymphoid follicles during relapses, and perforation of the intestine may occur then as in the primary infection, but it is much less common.

Site of Perforation.—The commonest site for perforation to occur is in the ileum, in its last few feet, but it has been found in other parts of the intestine in the following order of frequency, ascending colon, transverse colon, descending colon, pelvic colon and rectum, and also in the appendix, Meckel's diverticulum and jejunum. In the cases under review the majority of the perforations were in the ileum (see Table IV) in its last two feet; but two of them were at the ileo-cæcal junction and are best described as ileo-cæcal. The proportion of ileal perforations was by no means so great as is usual in typhoid, for as a

rule there are about five cases of ileal perforation to every one in the rest of the intestinal tract. There is more lymphoid tissue in the lower part of the ileum, and here as a rule the lesions of true typhoid are most marked. In paratyphoid fever, on the other hand, the large intestine is involved to a greater extent and is more severely affected than is usual in true typhoid, and in three out of five cases of perforation in paratyphoid the perforation was in the large bowel.

TABLE IV

Disease.	Last two feet of ileum.	Ileo-Cæcal.	Cæcum.	Appendix.	Descending Colon.	Pelvic Colon.
Typhoid . . .	5**	2*	1	1*	—	1
Paratyphoid A . .	1	—	—	1	—	—
Paratyphoid B . .	1	—	—	—	1	1
Total . . .	7	2	1	2	1	2

* In this case there were two perforations.
** In one of the ileal cases there were two perforations.

Pathology.—Perforation of an intestinal ulcer in typhoid or paratyphoid fever occurs, as a rule, when the sloughs are separating, but before this process is complete. The perforation is nearly always small, and will often only admit a pin. It is very seldom of greater diameter than a pencil. Sometimes the peritoneal coat is included in the slough, when the perforation is large, and generally round or oval. In other cases a cribriform opening

is found which looks as if the peritoneal coat had been gradually eaten away by ulceration. In the very small perforations the appearance suggests that an ulcer with a thin base has given way at one point. In a few cases there is a ragged torn perforation, probably brought about by great distension of the gut, or violent peristalsis.

Multiple Perforations.—In nearly 90 per cent. of cases of typhoid perforation collected from various sources the opening was single. In two cases in this series there was more than one perforation. In one case of typhoid in which an ileo-cæcal perforation was closed at operation, another perforation was found after death, eight days later, in a typhoid ulcer at the proximal end of the appendix. In the second case, also of typhoid, three perforations of the ileum were found at post-mortem examination.

Localisation of Infection.—Occurring most frequently in the ileum and opposite the mesenteric attachment, perforation of an intestinal ulcer is almost certain to cause a general infection of the peritoneum. In rare instances we find protective plastic peritonitis preventing leakage of intestinal contents, or if leakage does take place, the infection may be localised by adhesions, an occurrence which is, however, quite exceptional. A localised abscess may result in this way and also from leakage between the layers of the mesentery; or in the case of perforation of the large intestine the infection may be extraperitoneal, and again

with escape of intestinal contents a localised abscess may be formed.

Amongst John Hunter's specimens is one which illustrates the protection so rarely afforded by peritoneal adhesions to the base of a typhoid ulcer about to perforate.

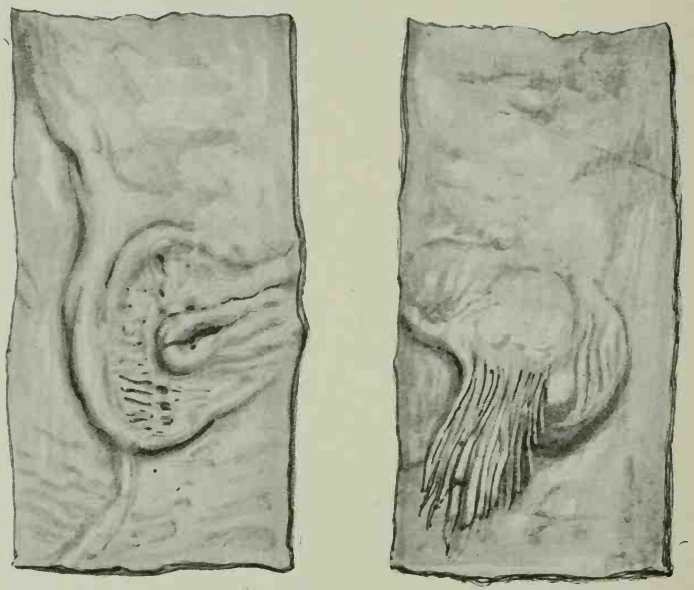


FIG. 22. Portion of ileum in which there is a small deep typhoid ulcer protected on the external surface by a peritoneal adhesion. (From Hunter's original collection. Museum of the Royal College of Surgeons of England. Specimen No. 2513.)

Two cases of typhoid perforation in the present series in their post-mortem appearances showed signs of an attempt by nature to prevent or limit infection of the peritoneum. In one case of perforation at the ileo-cæcal junction there was plastic peritonitis in the neighbourhood and adhesion of the omen-

tum. In another patient who died with symptoms of intestinal obstruction, the possibility of which must be borne in mind after typhoid and paratyphoid fever, a peritoneal band was found obstructing the ileum. The starting-point of the band was a small perforation in the last foot of the ileum, to which the omentum had become adherent. (This case is included in Table III, page 62.)

Symptoms. — When perforation occurs, the symptoms may be obvious or obscure. In the obvious cases, which are rare, there may be all or most of the classical symptoms and signs of an acute surgical abdominal emergency. The obscure and difficult cases are much the commoner, for with the usual small perforation in a patient whose sensations are blunted by toxæmia, any of the symptoms and signs may be slight or even absent.

In about three-quarters of the cases there is abdominal pain, generally severe, starting suddenly and persisting. It should be borne in mind, however, that abdominal pain occurs in about one-third of the cases of the typhoid group of fevers at some time during the course of the disease, but it is generally due to some complication, which may be abdominal, such as perforation of the intestine or gall-bladder, appendicitis, intestinal hæmorrhage, or cholecystitis; or extra-abdominal, such as phlebitis of the iliac vein, pneumonia or pleurisy. In perforation the pain is often general, and may be complained of in any part of the abdomen, but is

referred most commonly to the right iliac or umbilical regions. Some restriction of abdominal respiratory movement generally occurs. Tenderness and rigidity or increased resistance, especially of the rectus, is a very constant and important sign. Tenderness on rectal examination is also frequently found. Changes in liver dullness are too deceptive to be of any real value in making a diagnosis. Vomiting may occur, but usually is rather a late symptom. The patient, as a rule, becomes restless and distressed, while the characteristic facies often develops. There may be some increased distension of the abdomen, though it occasionally becomes retracted and rigid. Some change of temperature generally occurs. There may be a sudden fall, but just as often a rise of temperature is noted, perhaps with a rigor, and quite frequently there is no remarkable change. In three cases of the present series there was a fall, in five there was a distinct rise, and in six there was no notable change in the temperature. The pulse always becomes more rapid, and loses quality. In case of doubt, if time permits, a blood count should be made, and may help, for within a few hours of perforation there is a leucocytosis with increase in the number of polymorphonuclear cells. A blood count will be especially useful if repeated observations have been made previously, for then smaller changes will be appreciated. The same remarks apply to changes in the blood pressure.

Diagnosis.—With regard to the diagnosis of

perforation of the intestine we may begin by admitting that in rare instances, in a profoundly toxæmic patient, this accident may happen, and be unsuspected until revealed at post-mortem examination; also that during the course of typhoid or paratyphoid fever symptoms may arise which are so suggestive of perforation of a hollow viscus as to lead to operation on that supposition, when no such accident has occurred. It follows, therefore, that a diagnosis of perforation cannot be accepted unless confirmed either by operation or post-mortem examination.

In no case in this series was laparotomy performed without an acute lesion requiring immediate surgical intervention being found at operation, such, for example, as peritonitis, appendicitis, or perforation of the intestine. Many patients, however, have given us anxiety by presenting symptoms suggestive of perforation when their condition has been due to some comparatively trivial disturbance or the onset of some other complication, such as a mild appendicitis or thrombosis of the iliac vein.

It is especially important to be on the look out for perforation of the intestine in severe cases, during and after the second week, and more so in those who have marked tympanites or diarrhœa or have suffered or are suffering from intestinal hæmorrhage. The increased likelihood of perforation in cases with hæmorrhage is laid stress on by many authorities. The association of perforation

with hæmorrhage in the present series is shown in Table V.

TABLE V

Disease.	Number of cases.	Hæmorrhage.	Perforation.	Both hæmorrhage and perforation.
Typhoid . . .	1,118	50	9	3
Paratyphoid A . .	344	1	2	—
Paratyphoid B . .	1,038	16	3	1
Total . . .	2,500	67	14	4

Hæmorrhage was followed by perforation in a sufficient number of cases (nearly a third) to emphasise the necessity for a specially careful watch being kept on the progress of patients who have suffered from intestinal hæmorrhage. Perforation may take place at the same time as hæmorrhage, and the occurrence of much abdominal pain with intestinal hæmorrhage should arouse a strong suspicion of perforation.

Since early diagnosis is essential to success in treatment, it is imperative that the surgeon should have the opportunity of seeing the patient as soon as there is the slightest suspicion of perforation having occurred. It is no use waiting until symptoms and signs are well established, for then it may be too late and the case past hope.

Delays have dangerous ends, so much so that in a doubtful case the abdomen should be explored. No harm is likely to be done, and life may be saved. It is far better to open the abdomen and find no perforation than for a post-mortem

examination to reveal a perforation which could have been closed, perhaps with success.

Treatment.—It is interesting to recall that John Hunter wrote: "In whatever light we consider an inflammation of the peritoneum that is capable of producing suppuration, it is one of the most dangerous diseases we can meet with. How far in such cases it might appear desirable to make an opening in the abdomen, and throw in warm water repeatedly to wash out the matter, I will not undertake at present to determine."

In 1884 Von Leyden recommended the surgical treatment of intestinal perforation, and the same year Mikulicz reported the case of a man aged forty with typhoid perforation treated successfully by operation. The first successful case recorded in England was operated on by Bowlby in 1896. It is now universally accepted that the abdomen should be opened when a diagnosis of intestinal perforation is made, provided the case is not past hope. If there is the smallest chance of success, operation should be performed. Practically the only alternative to operation is the patient's death.

After the initial shock has passed off, the sooner the operation is done the better. After operation has been decided on, morphia may be given, but on no account should it be given before this stage. The operation can be carried out under local anæsthesia, and in some cases this will be quite satisfactory. A combination of local analgesia with gas and oxygen or small doses of ether inhalation to

keep the patient drowsy is, I consider, the best anæsthetic to use. An incision through the right rectus muscle is favoured by some surgeons, but though an incision on the right side may be convenient for dealing with a perforation in the usual site—the last portion of the ileum near the cæcum—it may be extremely inconvenient in the case of perforation of some parts of the colon. As we have seen, the large intestine is more likely to be the seat of perforation in paratyphoid fever, so that in this disease, at any rate, and probably as a general rule, a mid-line incision will prove the most suitable as a routine procedure. In some cases there may be special symptoms or signs, such as localised pain, tenderness, or rigidity, which will give some indication as to the most likely site of the perforation, and the most convenient incision for dealing with the condition.

In the vast majority of cases the opening is in the last two feet of the ileum, and an examination of this part of the intestine, beginning at the cæcum, will as a rule soon expose the lesion. The opening is closed, and in some cases it is well to take a leaf out of nature's book and stitch the omentum lightly over the line of suture to reinforce the weakened gut. If there is much distension of the intestine, it may be relieved to some extent through the perforation before it is closed. The peritoneal cavity is then emptied of fluid and escaped intestinal contents and very gently cleansed. It is necessary to examine for other perforations or

for ulcers in which perforation may be imminent. If there is a second opening, it is practically always near the first, so the search need not be extensive, and involves little expenditure of time. It is well to examine the appendix, and if necessary to remove it before closing the abdomen. It should also be remembered that the gall-bladder may perforate at the same time as the intestine. Speed is essential, and no time must be wasted. When the appendix or Meckel's diverticulum is the site of perforation, the best course will be to resect it.

Sometimes, with intestinal atony or paresis and symptoms of pseudo-ileus, instead of closing the perforation it is a wise procedure to perform an enterostomy, or in the case of a perforation of the appendix or Meckel's diverticulum the stump may be fixed in the wound instead of being closed. The value of intestinal drainage in such conditions has been emphasised in recent years. The patient is seldom in a condition to stand a resection, even if indicated by local conditions. In the very worst cases the surgeon may be content with simply draining the peritoneal cavity, by which means good results have been obtained in rare instances. Drainage is practically always necessary. Healing of the abdominal wall is often slow and difficult, and the sutures should be of non-absorbable material and should not be removed too soon.

Prognosis.— The prognosis depends largely on the stage of the typhoid or paratyphoid fever, on the time that elapses between the occurrence of

perforation and treatment by operation, and on the site of perforation. When perforation occurs during the stage of defervescence, the prognosis is better than when it occurs during the height of the fever. Operation within twelve hours of the perforation gives the best results. Perforation of the large intestine is a more serious accident than perforation of the ileum on account of the greater degree of sepsis, a factor which influences the mortality in other surgical affections of the large bowel. With peritonitis due mainly to pyogenic organisms the prognosis is much more grave than when the predominating bacteria are those of the typhoid group. Other factors which affect the prognosis are the state of distension of the bowel, the size of the opening, and the presence or absence of adhesions.

In the present series of cases an unusually large proportion of perforations occurred in the large intestine (see Table IV, page 64). The patients were in a very critical condition when operation was undertaken, often rather as a *dernier ressort* than with much expectation of success. Of the six cases submitted to operation, three were suffering from typhoid. The first of these perforated on the 17th day, an ileo-cæcal perforation being closed. Death occurred eight days later, and another perforation was found post-mortem at the base of the appendix. The second case perforated on the 70th day during a relapse complicated by broncho-pneumonia. The patient died twelve hours after operation. The third typhoid patient, who also had

broncho-pneumonia, perforated on the 31st day, and died three days later. In the patient with paratyphoid A, perforation occurred on the 30th day, and at operation an ileal perforation was closed with good result. In the cases with paratyphoid B, one patient, who was extremely toxic from the first, had a perforation of the sigmoid flexure on the 23rd day, and only survived operation thirty-six hours. In the other case of paratyphoid B an ileal perforation was closed on the 12th day, but death occurred six days later from peritonitis. In such a small number of cases the results, if good, may represent too promising a picture; while if the mortality is high, the results may appear too discouraging.

In the typhoid cases there was no recovery, but in paratyphoid one out of three recovered. That good results are to be obtained with promising cases under favourable conditions is clear from the statistics of the Royal Victoria and Montreal General Hospitals, where 140 perforations were closed with thirty-eight recoveries, or 27·14 per cent.; while from January 1909 to June 1914 Dr. G. E. Armstrong (*The Lancet*, August 1st, 1914) closed twenty-two typhoid perforations with only eleven deaths, a recovery rate of 50 per cent. The essential factors in obtaining good results are early diagnosis and prompt operation. The surgeon must be called in when there is the slightest suspicion of perforation having occurred, and there must be the closest co-operation between physician and surgeon during any such period of

anxiety. In diagnosis and treatment, as well as during operation, there is no time to spare.

Appendicitis

The vermiform appendix may figure in the typhoid group of fevers in various ways. Firstly, there may be difficulty in diagnosing between appendicitis and typhoid or paratyphoid fever; secondly, during the course of typhoid or paratyphoid the lymphoid tissue of the appendix is often involved in the ulceration, and perforation may take place through the appendix (Fig. 23), and, thirdly, an acute, subacute, or recurrent attack of non-typhoid appendicitis may supervene as a complication.

The diagnosis of typhoid or paratyphoid fever may be extremely difficult during the first few days, and when the onset is sudden and associated with severe abdominal pain, referred as it often is to the right iliac fossa, the simulation of appendicitis is very close. So close may the resemblance be, that it is not uncommon for such a case to be operated on. In the present series one patient with paratyphoid A was operated on as a case of appendicitis before his admission to the Isolation Hospital; and one patient who was proved later to be suffering from typhoid was explored on a diagnosis of appendicitis after his admission. The resemblance is more likely to lead to an erroneous diagnosis of appendicitis under peace conditions when typhoid is a comparatively rare disease. Under war conditions, when a medical

officer is confronted with a patient with abdominal symptoms, his mental bias leans more in the direction of typhoid or paratyphoid infection, with the result that during the two years under review twelve patients were sent into the Isolation Hospital as suspected enteric when they were really suffering from appendicitis.

With careful examination the mistake will seldom

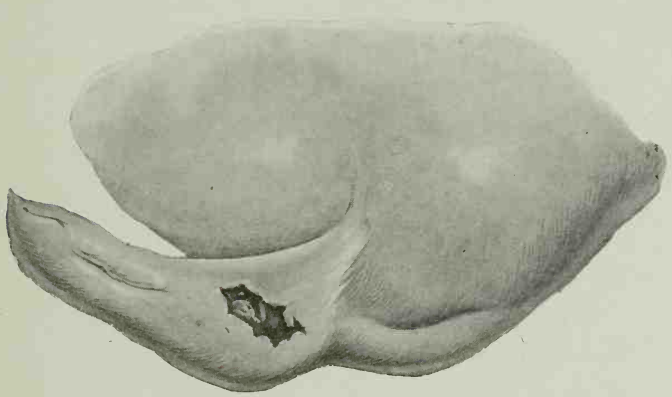


FIG. 23. *Paratyphoid* A. Ulcer of the appendix with a large perforation; the appendicular tissue around the ulcer was apparently healthy. (From *The Quarterly Journal of Medicine.*)

be made, for in typhoid or paratyphoid fever, even if pain be referred to the right iliac fossa, it is not associated with rigidity, tenderness, and rapid pulse except when a complication is present. In case of doubt, a blood count will be of value, and has settled the diagnosis more than once when there has been difficulty.

The involvement of the lymphoid tissue of the appendix in the typhoid lesions is by no means rare. In typhoid fever the large intestine is affected more

frequently than is generally supposed, while in paratyphoid it is the rule on post-mortem examination to find considerable ulceration of the cæcum, appendix, and colon. In the present series perforation took place through the appendix in one case of typhoid and in one case of paratyphoid A. (Fig. 23.)

The supervention of an acute, subacute, or recurrent attack of non-typhoid appendicitis in a patient with typhoid or paratyphoid fever is a serious complication, but remarkably rare considering the acute inflammation in the lymphoid tissue of the intestine and often of the appendix itself.

When the complication does arise, it is not always easy to decide on the best method of treatment. While there may be considerable anxiety about undertaking an operation on a patient with typhoid or paratyphoid fever, if acutely ill, there is also the fear that if operation be postponed the consequences may be serious. Fortunately the complication is not only rare, but in many cases subsides without operation. Much will depend on the condition of the patient. If not likely to be materially upset by the operation, it may well be performed, but if seriously ill and toxæmic, it is as a rule better to wait, unless operation is obviously required immediately. In the present series appendicitis supervened eleven times, and the only cases which proved severe were four which occurred during paratyphoid B. In one, a severely toxic case with petechial hæmorrhages, symptoms and

signs of appendicitis appeared towards the end, and a gangrenous appendix was found post-mortem. The three others were operated on, two of them recovering. In one an appendicitic abscess was drained and the appendix removed; in the second a gangrenous appendix was removed during convalescence. The third, jaundiced at the time of operation for appendix abscess, only improved for a time, and died with multiple abscesses in the liver, abscess of the lung and empyema. Bacillus paratyphosus B was recovered from the pus in the appendix region and also from the liver abscesses.

Peritonitis from other Causes

Other causes of peritonitis in typhoid and paratyphoid fever, besides perforation of an intestinal ulcer and appendicitis, are perforation of the gall-bladder, rupture of a liver abscess, and rupture of the spleen or of a splenic abscess. To these I will return when dealing with complications involving these organs.

Peritonitis has also been described as arising from extension of an abscess of the abdominal wall, from some coincident intestinal or pelvic conditions or from suppurating mesenteric glands. In only three cases of the present series was pus found in the mesenteric glands. Two of them were typhoid and one was paratyphoid B, but in none of them did the glands give rise to peritonitis.

In large collections of typhoid cases there are, as a rule, one or two cases of peritonitis for which no cause has been ascertained, and in the present series there was one fatal case of paratyphoid B in a non-inoculated man, on whom laparotomy was performed on the 16th day of his disease, when purulent peritonitis was found, but no perforation. At post-mortem examination no opening in the intestine and no other obvious cause for the peritonitis was discovered, but there was some adhesion between coils of intestine with purulent organised lymph. The investigation of such a case must include a careful search for suppurating mesenteric glands, a minute survey of the intestinal tract for a small opening or fissure or signs of plastic peritonitis over some deep ulcer, while the condition of the gall-bladder and appendix must also be examined.

It is generally accepted that peritonitis does occasionally arise from passage of infection through the peritoneal coat of the bowel, but fortunately it is of very rare occurrence. One of the most striking features of the typhoid intestine is the way in which ulceration frequently extends to the peritoneal coat and yet no trace of inflammation is apparent on the peritoneal surface of the intestine, contrary to the usual rule in other bacterial invasions of the intestinal wall.

The fact remains that there are certain cases of peritonitis in the typhoid group of fevers for which no local cause can be found, and infection

must have reached the peritoneal cavity owing to the general septicæmia, or have passed through the peritoneal coat of the intestine without any obvious perforation being present.

Subphrenic Abscess is met with occasionally, but in the present series there was no instance, though I have operated on two cases since this analysis was made. In each case the abscess was behind the liver, but the primary cause was not ascertained. The appendix is usually the part of the intestinal tract primarily responsible for the formation of subphrenic collections of pus on the right side.

Intestinal Obstruction.—Volvulus, intussusception, and other conditions leading to intestinal obstruction arise from time to time during the course of typhoid group infections, but intestinal obstruction may also develop as the direct result of the typhoid lesions in the intestinal tract. The formation of adhesions has already been described, and there are several cases on record of these adhesions giving rise to bands which have caused obstruction of the intestine, either during the course of the illness or at a later date. There was one instance amongst the non-inoculated typhoid cases in the present series. The patient was extremely ill, and developed symptoms of acute intestinal obstruction on the 40th day of the disease. The ileum was found to be obstructed by a band about twelve inches above the cæcum. The band ran from near the cæcum to the sacral promontory, and the starting-point was a small perforation in the

terminal portion of the ileum to which the omentum had become adherent.

It is stated that typhoid enteritis may also lead to intestinal obstruction by producing a stricture of the intestine in the process of cicatrisation of an ulcer. It is usual for typhoid ulcers to heal without any interference with the lumen of the bowel, but in rare instances bands in the submucous tissue have been described. They probably only occur when an ulcer is due to mixed infection, but cases of obstruction from this cause are reported to have followed attacks of typhoid fever. Though there are a few cases which appear to be definite examples of stricture after typhoid fever, there are, as stated by Treves (*Intestinal Obstruction*, Cassell & Co.), many reported cases which do not bear examination, and the association of a previous attack of typhoid fever with these examples is probably accidental.

CHAPTER V

THE SPLEEN

THE spleen is palpable in over 70 per cent. of cases of typhoid (McCrae), and either palpable or enlarged to percussion in nearly 60 per cent. of cases of paratyphoid fever (Torrens & Whittington). This organ forms a prominent feature of the clinical picture, and plays an important part in the pathology of the typhoid group of fevers, but is seldom the seat of surgical complications. So uncommon are surgical conditions and so difficult are they to diagnose that they are often more of pathological than of surgical interest. Still, operative intervention is sometimes required for conditions arising in the spleen during both typhoid and paratyphoid fever.

Rupture of the Spleen.—That rare accident, spontaneous rupture of the spleen, has occasionally been observed in the course of typhoid fever without any co-existent or previous malarial infection, and without an abscess being present. The tear may involve the capsule only, when it is of comparatively minor importance since hæmorrhage may not result; but if the laceration extends into the sub-

stance of the spleen, hæmorrhage is severe. In face of such an accident, the only hope lies in splenectomy, an extremely serious undertaking in a patient with typhoid or paratyphoid fever, and so far not attended with success.

Splenic Abscess.—Splenic abscess is met with occasionally in typhoid and paratyphoid fever, and is practically always, if not absolutely always, the result of infarction. When it is remembered how constantly foci of necrosis are found on examination of the typhoid spleen, and that the bacilli of these fevers can always be recovered from this organ in fatal cases, the rare occurrence of abscess of the spleen is rather striking.

In the present series there were three cases of splenic abscess, one in the course of typhoid (an inoculated man) and two in paratyphoid B, neither of them inoculated. The patient with typhoid who died on the 22nd day had pneumonia also, and was in a serious toxic condition throughout his illness. At autopsy suppurating infarcts were found in the spleen with an abscess on the surface. Bacillus typhosus was grown from the pus.

In one of the paratyphoid cases, which proved fatal from perforation of the sigmoid flexure on the 23rd day, two abscesses were found in the spleen on post-mortem examination, each being the size of a pigeon's egg. Bacillus paratyphosus B was grown from the pus. In the other case there were signs of trouble in the splenic region before death, for on

PLATE I.

Spleen. Paratyphoid B: The spleen shows a large abscess cavity at its lower pole, and along the anterior margin a small cavity has been opened. Between these two cavities the spleen is mottled yellow and blue-grey, and indicates an underlying third abscess. The wall of the lower abscess is adherent to the colon. (From *The Quarterly Journal of Medicine.*)

the 22nd day the patient complained of pain in the lower part of the left side of the chest on taking a deep breath. This passed off, however, and there was improvement in his general condition until the 34th day, when he vomited and his temperature rose to 102·6°F. and his pulse rate to 128 per minute. Later he complained of pain in the left hypochondriac region, and the abdomen became tender and rigid. His general condition became rapidly worse, and he died on the 36th day. At autopsy the intestine, including the appendix, was normal in appearance. The spleen (Plate I.) was found to be adherent to the parietes, to the colon, and to other viscera. There was an abscess the size of a plover's egg in the lower pole, and there were other smaller abscesses towards the hilum of the organ. Bacillus paratyphosus B was isolated from the pus obtained from these abscesses.

None of the cases were operated on for splenic abscess, and with the circumstances generally unfavourable—multiple abscesses, no definite diagnosis, and critical condition of the patients—operative treatment would probably not have met with any success.

Diagnosis.—It is not often that a diagnosis will be made with sufficient exactness to warrant surgical intervention in the early stages. Later a left-sided pleurisy or rapid enlargement of the spleen, or signs of subphrenic abscess may give some more definite indication of the condition, and the abscess may point or lead to œdema and redness over the

lower ribs. When, however, there is no definite indication of the condition, but a strong suspicion of splenic abscess exists, exploratory puncture may be justified and may give conclusive information. If puncture is decided on, the surgeon must be prepared to proceed to operation in case of a positive finding. A fine long needle is used with a syringe, and the puncture is made in the tenth intercostal space in the mid-axillary line. The patient is recumbent as a rule, but the puncture may be made with the patient in a sitting position, with the left arm raised. He should be instructed to hold his breath, and the aspiration should be carried out quickly. In case the patient makes any respiratory movements, the needle should not be fixed, but should simply be supported and allowed to follow the movements of the spleen.

Treatment.—Abscess of the spleen may be incised and drained, either from in front or from the side according to indications, and usually the spleen is found to be adherent to the parietes. Splenectomy is the operation of choice in some cases, and in those of the present series it was the only procedure which would have offered much prospect of success. It is said that with operation the prognosis is good, but operation is only undertaken when circumstances are favourable: good condition of the patient (sometimes an abscess develops during convalescence), a single abscess, and adhesion to the parietes.

From the surgical point of view I think the most

interesting points about the spleen in typhoid and paratyphoid fevers are its rôle in the general pathology of the disease, its relation to infection of the biliary passages, and to the persistence of typhoid group bacilli in the body, to which I will return when discussing the Carrier Problem (page 167).

CHAPTER VI

THE LIVER, BILIARY PASSAGES, AND PANCREAS

Liver Abscess. *Pathology.*—The liver in fatal cases of typhoid and paratyphoid fevers shows, as a rule, parenchymatous degeneration and foci of necrosis, but abscess in this organ is extremely rare.

The elimination of micro-organisms by the liver from the portal circulation and their excretion with the bile goes on constantly during the course of these fevers, but the biliary tract, as a rule, escapes any obvious inflammatory reaction. Sometimes, however, the bile ducts may be affected by an acute suppurative process and liver abscess may develop in this way. The more usual routes of infection of the liver are through the hepatic artery or the portal vein. When the infection is carried by the hepatic artery, the liver abscess is part of a systemic pyæmia, with abscesses in other parts of the body. When the portal vein is the channel of infection, there is generally a gangrenous inflammation of some part of the intestinal tract (nearly always the appendix), with thrombosis of the veins in the neighbourhood. Abscesses of the liver in these fevers are more often multiple than single, and

surgical intervention seldom leads to a successful result.

Sometimes, however, a single abscess develops, either being single from the first, or being formed by the coalescence of more than one. Drainage can then be provided and the prognosis is not unfavourable. In cases of multiple abscesses surgery offers no prospect of relief, though improvement is said to have followed drainage of the gall-bladder.

Symptoms, Diagnosis, and Treatment.—The main symptom complained of is pain in the region of the liver, and there is generally some appreciable enlargement of this organ. In some cases there is jaundice, and often the patients develop that curious earthy colour associated especially with liver abscess. There is usually an increasing leucocytosis. If liver abscess is suspected, exploratory puncture should be carried out under general anæsthesia, and the surgeon must be prepared to proceed to operation if necessary. A medium or full-sized needle should be used with the aspirator. If there is any local sign of the situation of the abscess, exploratory puncture should be made first in that direction, but if not the puncture may be made in the anterior axillary line in the eighth or ninth intercostal space, the needle being directed inwards, upwards, and backwards. The liver may also be explored by punctures in the ninth or tenth space in the mid-axillary line, or below the angle of the scapula in the tenth intercostal space. The needle may be

introduced for four or five inches, and if no pus is found at first, the liver should be explored systematically by five or six more punctures. The pus from liver abscesses is often dark brown in colour, and care must be taken not to overlook a positive finding by mistaking pus for blood. The abscess may be opened and drained either from in front or behind, the decision as to site of operation depending on the situation of the abscess.

In the present series there were two cases of liver abscess; both occurred in paratyphoid B. One of them died from portal pyæmia thirty days after operation for gangrenous appendicitis with abscess in the iliac fossa. Multiple abscesses were found in the liver, and the pus yielded a pure culture of bacillus paratyphosus B.

The second case presented many points of interest. The illness *began* with pain in the right shoulder. He "reported sick" after four days' malaise, and was admitted to hospital as a case of "myalgia and debility." Two days later his temperature began to rise, and later he had some bilious vomiting. On the 20th day of his illness he was admitted to the Isolation Hospital, when by agglutination tests he was diagnosed as suffering from paratyphoid B. His main symptoms were pain in the right shoulder and vomiting. On percussion over the base of the right lung resonance was impaired. A focus of suppuration was suspected soon after he came under observation, as his blood count showed a leucocytosis of 16,000, with a high percentage of poly-

morphonuclear cells. The severity of the pain in the shoulder increased, vomiting persisted, the dullness at the base of the right lung extended upwards about an inch and a half, and the blood picture became more and more indicative of abscess formation (see Table VI).

TABLE VI

—	Polymorpho-nuclear cells.	Mononuclear Leucocytes.	Lymphocytes.
22nd Day . . .	78·2%	10·6%	10·6%
25th Day . .	81·1%	7·5%	11·4%
31st Day . . .	85·0%	5·9%	9·1%

A trace of bile was found in the urine, and there was some slight tenderness and increased muscular resistance below the ninth costal cartilage on the right side. The suppurating focus was evidently in the liver, and although pus could not be obtained on exploratory puncture, operation was decided on. The abscess, however, ruptured spontaneously on the 31st day of the disease, and the pus was discharged through a bronchus, after which rapid improvement took place, and the soldier eventually returned to duty. Short chain streptococci and diplococci were recovered from the pus expectorated. It has been estimated that over 25 per cent. of liver abscesses rupture spontaneously, and that more than half of these do so in an upward direction.

The Gall-Bladder and Biliary Passages

Pathology.—Pure cultures of typhoid and paratyphoid bacilli are obtained almost constantly from the contents of the gall-bladder in fatal cases of these diseases, and with more certainty than from almost any part of the body except the spleen and possibly the bone-marrow. The bacilli may still be found in the bile months and even many years after the patient is well, when they may or may not give rise to cholecystitis and gall-stones. The bacilli may be present in the biliary tract without the patient ever having had symptoms of typhoid or paratyphoid fever, and an acute cholecystitis may be the sole manifestation of infection with typhoid or paratyphoid bacilli.

The organisms reach the gall-bladder in large numbers through the bile, for the liver eliminates them from the blood carried to it by the portal vein from the gastro-intestinal tract and spleen. The liver does not succeed in killing all the organisms brought to it, possibly because it is often itself the seat of parenchymatous degeneration. The gall-bladder may also be infected through its arterial blood supply, or possibly by ascending infection from the intestine, but the latter idea is now practically abandoned. The constant presence of the bacilli in the bile in the course of these fevers provides a possible mode of infection even long after they have disappeared from the systemic circulation. As a rule the passage of these micro-

organisms along the biliary tract gives rise to no symptoms or signs of inflammatory reaction, and no apparent pathological changes, just as the urinary tract may not be obviously affected by a typhoid or paratyphoid bacilluria.

Acute Cholecystitis occurs occasionally, however, in the course of these fevers. It is generally catarrhal in character, but may be suppurative and lead to empyema of the gall-bladder, or may go on to ulceration or even to perforation. In these cases there is some question whether the infection carried by the bile leads to the ulceration and then occasionally to perforation, or whether the gall-bladder wall is infected by the blood stream and the ulceration is secondary to the formation of abscesses in the gall-bladder wall. Probably the condition arises as a result of both methods of invasion, for bacilli and abscesses have frequently been found in the wall of the gall-bladder in these cases. Cholecystitis in typhoid and paratyphoid fever is generally but not always due to the typhoid group of bacilli. Bacillus coli communis has also been found either in pure culture or mixed with other organisms.

Symptoms and Diagnosis.—Though a mild cholecystitis probably occurs in a fair percentage of cases of typhoid and paratyphoid fever, it is only possible to diagnose the condition with any degree of certainty in a few. With the sudden onset of pain in the right hypochondrium with tenderness, muscular resistance to pressure over the gall-bladder region,

perhaps some rise of temperature and leucocytosis, and often some enlargement of the ll-bladder which may become palpable, a diagnosis of cholecystitis may be made. Jaundice may also be a symptom, and was present in half the cases of cholecystitis in the present series. Jaundice was noted in over 2 per cent. of all the cases of typhoid and paratyphoid fever of this series, but it is not necessarily indicative of cholecystitis, and as a rule it is catarrhal in character and of no immediate surgical significance.

Cholecystitis is commonest in young adults, but may also occur in children and in older people. The time of onset is generally about the third week or later, but it varies very widely in different cases, and may develop as early as the 8th day or not until convalescence. It may also occur as a sequel, and arise months or years after the attack of typhoid or paratyphoid fever. Also, as already mentioned, typhoid cholecystitis may be the sole manifestation of the disease, and typhoid or paratyphoid infection may be found when operating for gall-stones when there is no history of the patient ever having had typhoid or paratyphoid fever. It must be remembered, however, that in its mildest form an attack of one of these fevers may pass almost unnoticed or be unrecognised and classed as a minor ailment. The cholecystitis which occurs during the course of one of these fevers is seldom associated with cholelithiasis, but gall-stones are generally present in those cases which arise months

or years after an attack of typhoid or paratyphoid fever.

The symptoms of the condition usually persist for about a week and then subside, but in the rare cases in which suppuration or ulceration takes place symptoms persist or get progressively worse, when there is always the danger of perforation occurring. If stones are present in the gall-bladder before the patient is infected with typhoid or paratyphoid, there is a greater likelihood of cholecystitis developing, and of serious results supervening.

Treatment.—The treatment of cholecystitis arising as a complication of the typhoid group of fevers is expectant, but a very careful watch has to be kept on the patient, and the danger of perforation of the gall-bladder occurring must be remembered. Surgical intervention is called for if the symptoms are very severe and progressive, or if they do not subside. Urotropine has been shown to be a valuable antiseptic for the biliary as well as for the urinary tract, and may be given in doses of as much as twenty grains three times a day. Castellani states that urotropine is useful in preventing complications in the biliary tract, and it may be prescribed with advantage during the course of typhoid and paratyphoid fevers. If surgical intervention becomes necessary the operation decided on will depend to a large extent on the condition of the patient. If circumstances permit, the operation of choice is generally a cholecystectomy. Although this may be the ideal procedure in some cases, there

are others in which immediate relief must be given by a cholecystostomy, and the removal of the gall-bladder must be left until local conditions are more favourable and the patient better able to stand the shock of the bigger operation.

Perforation of the Gall-Bladder.—Perforation of the gall-bladder is a most serious, but fortunately a rare complication. It demands immediate laparotomy, and good results have been obtained by operation. As to the exact procedure to follow after opening the abdomen, here again the surgeon will be guided to a large extent by the general condition of the patient and the character of the local conditions found when the gall-bladder has been explored. In the present series there was no case of perforation of the gall-bladder.

It is interesting to record that John Hunter described the history and post-mortem appearances of a case of typhoid perforation of the gall-bladder in the same case as that from which the specimen shown in Fig. 13 was taken. "Winter, 1763–4.—A man in Mr. Fordyce's hospital had just recovered of a fever when he was taken ill with a violent pain in his belly just at the navel. He was ordered a clyster and a blister, but he had a natural stool; the pain continued, and within twenty hours of the attack he died. Upon opening the belly we found the peritoneum very much inflamed; the epiploon covered the whole (of the) viscera and was of a darkish colour as if wetted with blood. We found a universal inflammation in the whole visible

contents of the abdomen; the liver was glued to the diaphragm and the stomach wherever it touched. All the intestines were likewise glued together, and of a very dark colour where there was no compression by other surfaces. I examined the gall-bladder, and found it as large as my fist, and two holes at its fundus. Upon introducing my finger I found two more. There was but little bile in it, for it was got loose in the cavity of the abdomen, and had by that (means) tinged the glutine.

"I took out a piece of the ileum to inject to see the size of the vessels, which I did.

"Upon the inside of the intestine I observed several bodies, some as broad as a farthing, some smaller: they had a depression in their middles, so that they appeared like circular or oval ridges or risings. The texture of these risings was spongy or cellular, whose openings looked into the guts.

"Some of them appeared to be a thickening of the rugæ of the internal surface, for in the oval ones I could trace two rugæ into them. Why they should be depressed in the middle I can't tell, but this appearance I have often seen.

"Was the gall-bladder become rotten in this short time, or was it burst as the pain came on and was the cause of this universal inflammation in the abdomen? This is somewhat reasonable, as the inflammation seemed to be more on the external surface of the viscera than on the internal." (*Hunterian MS.: Dissections of Morbid Bodies*, No. 68, p. 94.)

TABLE VII

Disease.	Cases.	Liver abscess.	Chole-cystitis.	Gall-stones.	Jaundice.
Typhoid inoculated . .	821	—	4	1	19
Typhoid not inoculated .	297	—	2	—	8
Paratyphoid A inoculated .	123	—	—	—	3
Paratyphoid A not inoculated .	221	—	2	—	4
Paratyphoid B inoculated .	239	—	1	—	2
Paratyphoid B not inoculated .	799	2	6	1	17
Totals	2,500	2	15	2	53

Cases of Cholecystitis.—In the present series there were fifteen cases with more or less definite symptoms of cholecystitis. There was no case of perforation of the gall-bladder. In only one case was surgical intervention necessary, and, curiously enough, the patient was one of the few women in the series. (Out of 2,500 cases, only 14 patients were women.) The patient was 29 years of age. Three and a half years previously she had been operated on for appendicitis and an appendicitic abscess had been drained. She was admitted to hospital on December 16th, 1915, suffering from paratyphoid B, against which she had not been inoculated, and in the third week of her illness developed acute cholecystitis. The symptoms varied in severity from time to time, but never absolutely settled down. A swelling could be felt in the right hypochondrium, and there was considerable tenderness over the region of the gall-

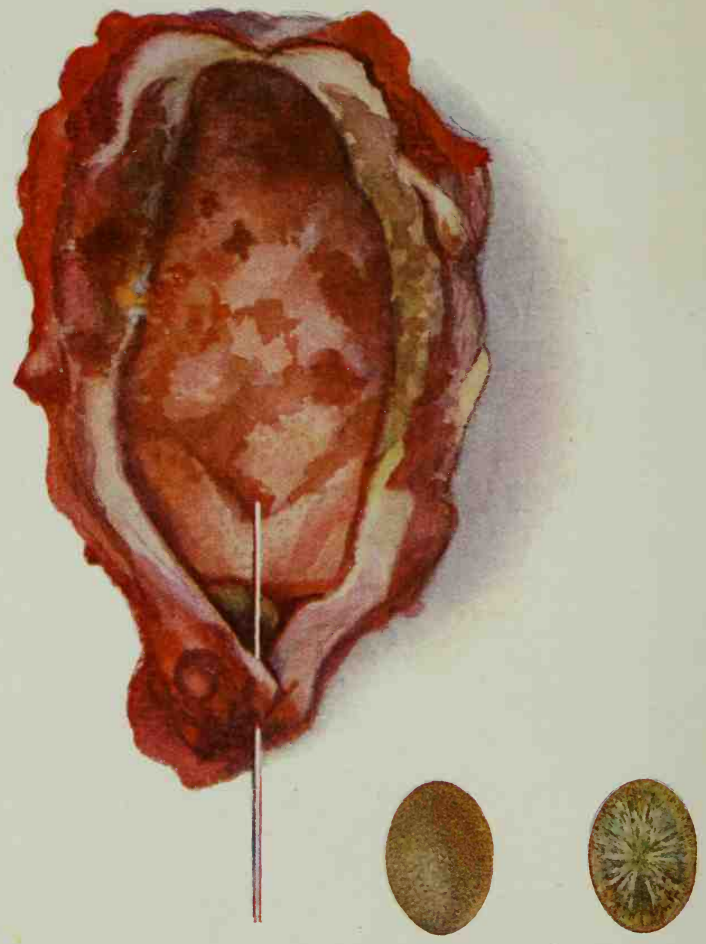


PLATE II.

Gall-bladder and gall-stone. Paratyphoid B: Acute cholecystitis with abscesses in the wall of the gall-bladder. The gall-stone may have been present before the attack of paratyphoid fever. (For this drawing I am indebted to Mrs. R. F. Bolt, a V.A.D. in the Hospital.)

bladder. Palpation of the swelling nearly always led to some rise of temperature. Cholecystectomy was performed on February 15th, 1916, and the patient made a good recovery. The gall-bladder was not enlarged. It contained turbid purulent bile-stained fluid, and there was a gall-stone the size of a nutmeg in the neck of the viscus. The omentum was adherent to the fundus, and in the wall nearest the liver was a diffuse infiltration with a small abscess communicating by an opening in the mucous membrane with the cavity of the gall-bladder (Plate II.).

The mucous membrane generally was acutely inflamed with many petechial hæmorrhages under its surface. Bacillus paratyphosus B was obtained in pure culture from the contents of the gall-bladder and from the abscess in its wall. The stone was a practically pure cholesterin calculus, but was not examined bacteriologically until later, when no micro-organisms were found. It could not therefore be said that this stone had been formed during the attack of paratyphoid fever, especially as the patient had suffered from a septic condition in the intestinal tract three and a half years previously. She had never had, however, any symptoms referred to the gall-bladder before the third week of her attack of paratyphoid. If the gall-stone was present before this illness, then it was probably a predisposing factor in causing the paratyphoid cholecystitis.

Relations of Typhoid Group Infection to Cholelithiasis.—There is every reason to believe that gall-stones may form very rapidly, and well within the sixty to seventy days which elapsed between the time of onset of paratyphoid and the time when operation was performed in this case. Mignot (*Arch. gén de méd.*, Paris, 1898, clxxxii. 129) induced the production of two cholesterin gall-stones in a dog in between five and six months. He infected the gall-bladder with bacillus coli communis, and later introduced a thread and suspended it from the fundus of the gall-bladder. The gall-stones were formed on the thread.

Hurst and Adams (*Proc. Roy. Soc. Med.*, 1910, iii. Clin. Sect. 169) reported the case of a woman, aged twenty-four, who had typhoid fever which began on December 10th, 1909, and at operation on February 18th next year (seventy days after the onset of the fever), twenty-five faceted cholesterin gall-stones, varying from $\frac{1}{8}$ to $\frac{1}{4}$ inch diameter, were found in the gall-bladder. Bacillus typhosus was obtained in pure culture from the fluid and pus in the gall-bladder, and from the centre of some of the stones. While many cases have been recorded of gall-stones following typhoid cholecystitis and of typhoid bacilli being found in them, Hurst and Adams ascertained that in only six were the stones found at periods of less than six months (five months, eight weeks, five weeks, five weeks, four weeks, and four weeks) after the onset of typhoid fever. In none of them were typhoid bacilli found

in the stones, and in only two were they reported as being obtained from the gall-bladder contents. In the present series there was a case of typhoid in which twenty-one gall-stones were found, post-mortem, in the gall-bladder of a man who died of intestinal hæmorrhage on the 17th day of the disease, but the stones may have been present before his attack of typhoid fever.

The important relation between typhoid group infection and the development of gall-stones has been established by many observations. Symptoms of gall-stones have been observed to follow typhoid fever; calculi have been found when operating on the gall-bladder after an attack of typhoid fever, whether after an interval of a few months or many years; a history of typhoid infection has been obtained in many cases of gall-stones; and the bacilli have been found in the fluid contents of the gall-bladder and in the stones themselves. We know, however, that bacilli pass along the biliary tract in all cases of typhoid group infection, but that gall-stones develop in only a few, so that it is not only the presence of the bacilli which determines the formation of gall-stones. In the great majority of cases the passage of the bacilli through the tract leads to no inflammatory reaction or to very little; in others it induces a mild catarrhal cholecystitis, and it is this which makes conditions favourable for the development of gall-stones.

This influence of the typhoid group of infections in determining a mild catarrh which favours the

production of gall-stones is of more than surgical interest, for it sets up local pathological conditions which may lead to persistence of the infection, and the specific bacteria have been isolated when operating for gall-stones many years after an attack of typhoid fever. There are other cases in which no gall-stones develop and no cholecystitis is present, but still the bacilli are present in the biliary tract. The question is, where do these bacilli come from and where have they been hiding and multiplying? Are we justified in locating their seat of vegetation in the biliary tract when, as far as can be ascertained, that tract is normal? The question is dealt with in the chapter on the Carrier Problem, where the suggestion is advanced that they multiply in the spleen and pass into the splenic vein and thence to the liver and biliary tract (page 167).

Pancreatitis.—Pancreatitis has been described in a few instances as a complication or sequel of typhoid fever, and the hæmorrhagic form has been met with. The infection will generally be dependent on an acute inflammatory condition in the biliary tract, and drainage of the gall-bladder may be of benefit. Moynihan operated on a boy with typhoid fever, and found the head and part of the body of the pancreas hard and tense, and twice its normal size. He drained the gall-bladder, and typhoid bacilli were found in the discharge for some weeks afterwards. Urotropine was given, and the discharge became free from bacilli.

CHAPTER VII

THE CARDIO-VASCULAR SYSTEM

During typhoid and paratyphoid fevers the cardio-vascular system is occasionally the seat of complications for the treatment of which surgical aid has to be invoked. Abscess in the wall of the heart and purulent pericarditis have been described, but may almost be classed as curiosities of surgical pathology. Thrombosis may occur in peripheral vessels—as a rule taking place in the veins where the circulation is more feeble and the blood more coagulable—but arterial thrombosis also arises in rare instances.

Gangrene — Arterial Thrombosis. — Thrombosis plays some part in the origin of bedsores and those cases of gangrene about the face and external genitals which occasionally occur in the course of these fevers. In the typhoid cases of this series there was one case of cancrum oris which proved fatal. Gangrene of an extremity is a well-known though rare complication which may exceptionally be the result of embolism, but in most cases is due to arterial thrombosis following an acute arteritis,

The vein may be thrombosed as well as the artery, and gangrene has developed after venous thrombosis only. Keen showed that, excluding bedsores, the legs, being distal parts of the circulatory system, suffered from gangrene more frequently than all other parts of the body put together, and that arterial obstruction arises as frequently in one lower limb as in the other. Amputation is necessary, and, as the gangrene is usually of the dry variety, a line of demarcation may be waited for, though in the event of the gangrene being moist and extending there is no justification for delay. In the present series this rare condition was not met with, but there were forty-two cases of venous thrombosis. (See Table VIII.)

Venous Thrombosis.—This complication, while not calling for surgical intervention during the course of the disease, except in the rare event of gangrene supervening, is nevertheless of considerable interest to the surgeon, for it may lead to serious disability. Not only is convalescence prolonged, but the circulation in the limb may be permanently damaged. Varix may develop or there may be a persistent tendency to œdema. This may be a source of annoyance and inconvenience, besides leading to some weakness and disability of the limb.

With obstruction of the external iliac vein there may be the additional inconvenience of large and tortuous veins developing in the abdominal wall to carry the venous return from the lower extremity. It is therefore of importance to give careful

attention to the causes, symptoms, prevention, and treatment of this complication.

TABLE VIII

Diseases.	Cases.	Thrombosis.	Per-centage.	Left side only.	Right side only.	Both sides.
Typhoid inoculated .	821	7	0·85	11	3	3
Typhoid not inoculated	297	10	3·36			
Paratyphoid A inoculated	123	—	—	2	—	—
Paratyphoid A not inoculated . . .	221	2	0·90			
Paratyphoid B inoculated	239	3	1·25	19	2	2
Paratyphoid B not inoculated . . .	799	20	2·50			
Totals . . .	2,500	42	1·68	32	5	5

The incidence of venous thrombosis in published series of typhoid cases is usually between 2 per cent. and 3 per cent. The percentages in the present series are shown in Table VIII, from which it will be seen that the complication was commonest in typhoid, fairly frequent in paratyphoid B, and less frequent in paratyphoid A. The femoral vein is the one most commonly affected, and in the present series (counting the 5 bilateral cases as 10) there were 25 instances of thrombosis in the femoral vein, 10 in the veins of the leg, 5 in the iliac, 4 in the popliteal, and 3 in the internal saphenous veins. There was no case of thrombosis in the upper limb, though this does occur sometimes. As usual, the *left lower limb* was affected in the great majority of the cases—a preponderance for which it seems

reasonable to accept an anatomical explanation, and one which affects the veins only, for, as already stated, arterial thrombosis occurs as frequently in one limb as in the other. Keen was convinced "that the obstruction to the return of venous blood by reason of the compression of the left common iliac vein, where it passes under the right common iliac artery, was the most potent factor in determining this preponderance." Mr. Warrington Haward accepts this view, and points out that "not only is the vein crossed by the right common iliac artery, but also by the left internal iliac artery as it passes downwards to the sacro-sciatic foramen, both of the arteries crossing the vein almost at a right angle, and in marked contrast to the relations on the other side" (*The Lancet*, 1906, vol. i., March 10th and 17th). The rectum, especially if loaded, may also have some influence in retarding the flow of blood through the left iliac veins, and constipation is not uncommonly a difficulty in the treatment of cases of typhoid group infection.

Time of Onset.—Thrombosis arises most frequently during the third and fourth weeks of the disease. In the present series the time of onset ranged from the 14th to the 80th day, the average date being the 27th day. Over half the cases began during the fourth week, about a quarter after that period, and the remainder in the third week. Since it generally arises after the acute stage of the disease is over, this complication does not as a rule seriously affect the patient's chances of recovery, but may in a

critical case turn the scale against him, or, in an exceptional case, directly lead to a fatal termination.

Of the patients of the present series, only one died. He was suffering from typhoid fever, proved by the cultivation of bacillus typhosus from his blood. He was seriously ill and in a very toxic condition. Thrombosis of the left femoral vein developed on the 23rd day, and he died on the 45th day. Post-mortem examination showed that thrombosis had contributed to the fatal result, for while the Peyer's patches and solitary follicles were pigmented black, with no evidence of existing ulceration, the left femoral and iliac veins contained firm thrombi, and there were many infarcts in the heart, lungs, and kidneys. The usual terminations are either disintegration of the clot, which is carried into the blood stream in minute particles without doing any harm, or the thrombus becomes "organised" by the gradual removal of the blood clot and its replacement by newly formed connective tissue.

Late Effects.—We have had no opportunity of seeing the after-effects of thrombosis in the present series of cases, but it was by no means uncommon, when examining reservists at the outbreak of war, to find persistent swelling of the limbs and varix, sometimes extending on to the abdomen, amongst those who had suffered during the South African campaign from typhoid fever complicated by thrombosis. The accompanying photograph is of an officer who, during an attack of typhoid fever, in March 1913, had thrombosis of the iliac vein. In

spite of careful treatment and twelve months' rest from work, he developed large varices, and is now incapacitated by them.

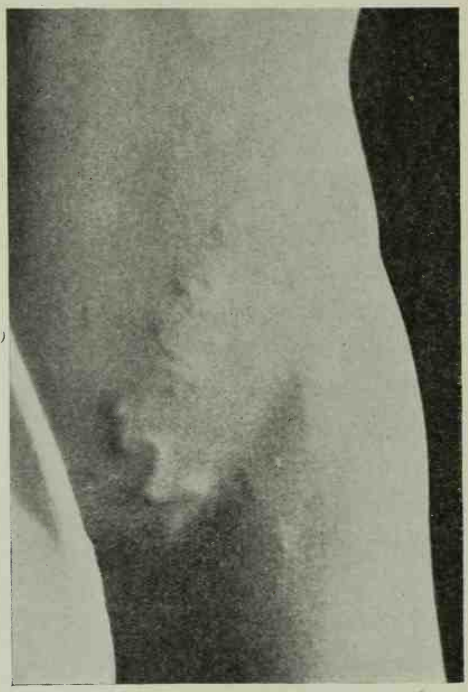


FIG. 23. Varicose veins. Enlargement of superficial veins to compensate for obstruction of the external iliac vein.

The subsequent condition was examined by McCrae in a series of patients who suffered from thrombosis during typhoid fever, and there was more or less disability in all of them.

Pathology.—The consideration of phlebitis and thrombosis takes our thoughts again to John Hunter, whose observations in 1784 first showed

"that the inside of veins as well as of all other cavities, is a seat of inflammation." The subject was dealt with very fully in 1906 by Mr. Warrington Haward, in whose Hunterian Lectures is given a complete account of the phenomena which lead to thrombosis within the living vessels. In typhoid and paratyphoid fevers various conditions exist which favour the occurrence of thrombosis. The heart's action is feeble and remarkably slow and the general *circulation* is sluggish, the nutrition of the *blood vessels* is impaired and the vessel walls infected, while the *blood* itself shows a greater tendency to coagulation. Any enfeeblement of the *circulation* or retardation of the blood current, which is slower, less forcible, and more easily retarded in the veins than in the arteries, makes thrombosis more likely not only by its effect on the blood, but also by its effect on the vessel wall. The *endothelial lining* is dependent for its nutrition on the blood within the vessel, and free circulation is as necessary for its welfare as for that of the blood. The other coats of the vessel are supplied by the vasa vasorum, and may also be affected by a sluggish circulation. Moreover, all layers of the *vessel wall* may suffer from the infection with micro-organisms. Bacillus typhosus has been found in the thrombi and the walls of the blood vessels. Other organisms, particularly staphylococci, have also been found. Phlebitis obviously precedes the thrombosis in a certain number of cases, and in the present series there were clear indications of this in nine cases, or

20 per cent. Probably it was an important factor in more, if not in all. The coagulability of *the blood* is increased during the third and fourth weeks of typhoid fever, the period when thrombosis generally occurs. Whatever divergence of opinion there may be as to the existence or not of the blood platelets as formed elements in the circulating blood, it is agreed that they fill a prominent rôle in the process of coagulation, and Osler's observation that in typhoid fever there is no increase of blood platelets during the first week, but a notable increase in the third and fourth weeks, may have an important bearing on the subject of thrombosis.

Changes in the chemical composition of the blood are by no means the least important of the influences leading to thrombosis. An addition to the amount of calcium salts in the blood markedly increases its coagulability, as shown by Wright, who also demonstrated that the presence of CO_2 in the blood has the same effect. In typhoid and paratyphoid fever the calcium salts in the blood may be increased by an injudicious persistence in a milk diet. The increase in the CO_2 content of venous blood may have a certain influence in increasing the tendency to thrombosis. With feeble circulation and often some lung complication and, beginning in the third week, some diminution of red cells and of hæmoglobin, the proper oxygenation of the blood may be interfered with and the CO_2 content increased in this group of diseases.

Symptoms.—The symptoms of venous thrombosis are generally clear and unmistakable. As a rule

the first symptom is pain which is felt in the course of the affected vessel. In the case of the veins of the leg, pain in the calf is usually complained of. There is some local tenderness, and in cases with definite phlebitis this is very marked, and associated with redness and swelling in the course of the vessel. There is nearly always some rise of temperature at the onset. Very often the temperature has already settled at about normal before thrombosis occurs, but even if this is not the case, any existing fever is generally increased by the onset of thrombosis. Quite commonly there is a rigor at the onset. There is generally some leucocytosis. At a later stage the thrombosed vein can be distinctly felt. Œdema appears at a variable time after the onset, sometimes being noticed very soon or even being the first sign to direct attention to the condition. In other cases œdema does not appear until after two or three days—suggesting that it has taken that time for the vein to become obstructed.

The sudden onset of thrombosis of the iliac veins, with abdominal pain associated with fever and leucocytosis, sometimes suggests the occurrence of some abdominal complication, such as perforation of the intestine or appendicitis. But, though the resemblance may be very close, there is not, as a rule, the same rigidity of the abdominal muscles, and if œdema is present, examination of the limb will soon give a clue to the correct diagnosis.

Prevention and Treatment.—While the main factor in the etiology of venous thrombosis is probably

infection with the typhoid or paratyphoid bacilli, and is therefore not directly under our control, we can influence certain of the contributing factors. By attention to these factors some cases can probably be prevented, and after the development of thrombosis much can be done to promote the absorption of the clot and the re-establishment of the normal circulation. If the normal blood channel is not reopened, then care in after-treatment may do something to prevent or mitigate the serious effects of the venous obstruction.

After the second week of typhoid and paratyphoid fever, and still more after the third week is past, *if the patient is progressing favourably to convalescence* the circulation may be stimulated, the patient may be encouraged to move his limbs, and gentle massage may be begun. Constipation should be avoided, and plenty of fresh air ensured. The diet should be light, and food rich in calcium salts should be avoided; milk should be citrated, and plenty of fluid with abundant citrates should be taken. It has been suggested that it is unwise to administer citrates or in any way to make the blood less coagulable in typhoid and paratyphoid fever from fear of increasing the likelihood of intestinal hæmorrhage. The essential factor in intestinal hæmorrhage, however, is not the condition of the blood, but the extent and depth of the ulceration. Moreover, hæmorrhage generally occurs before the third week. Only sixteen out of sixty-seven cases of intestinal hæmorrhage began after the third week in this series, and

then only in patients who were obviously not progressing steadily towards convalescence. Thrombosis, on the other hand, developed *after the third week* in three-fourths of the cases.

If symptoms of phlebitis arise before thrombosis is definitely established, the affected limb must be kept absolutely at rest, and if very painful should be wrapped in warm dry wool and the foot of the bed should be raised. A purgative should be given and citric acid prescribed. The diet must be light and plenty of citrated fluids should be administered. If the patient is too ill to be carried out into the fresh air, or to allow of much attention being given to his phlebitis, the administration of oxygen may be of benefit. When thrombosis is established, the same course should be followed. Great care must be taken to minimise movements and efforts, owing to the risk of detachment of clot. Easy movement of the bowels must be ensured, and if the patient has any respiratory trouble, the effort of coughing must be diminished as much as possible by the administration of suitable drugs.

Major H. Fairly Marris has had some successful results in venous thrombosis by the intravenous administration of sodium citrate (*Brit. Med. Journ.*, December 22nd, 1917, p. 822). He gave 10 ounces of a ·5 per cent. solution of sodium citrate in saline, and repeated the dose at intervals of a few days when necessary. The dose seems very small, and up to the present the cases have been too few to warrant any definite statement as to the advantages

of the intravenous method of administration, but some of the results have been very good.

A patient with thrombosis will have to remain in bed for several weeks. Six weeks is generally a safe period for rest, but if the circulation is soon re-established, movement may be begun correspondingly sooner. If the vein is permanently blocked or partially obstructed, efforts must be directed to preventing the undue dilatation of the superficial vessels and to encouraging the enlargement of the deeper collateral veins. This can be done by elastic supports, and the adoption of massage and muscular exercise as soon as considered safe. Bandages or other elastic supports should be worn by the patient for some months after he begins to walk, and may be required permanently.

CHAPTER VIII

THE URINARY SYSTEM

Pyelitis and Cystitis.—In a large number of cases bacilli of the typhoid group are found in the urine at some period of the disease, generally about the third week or later, or sometimes not until convalescence is established. In typhoid fever they are said to be found in the urine in about a quarter of the cases, but in paratyphoid the proportion has been less and in inoculated individuals it is smaller still.

Pathology.—It is generally accepted that in order to allow the passage of micro-organisms it is necessary for the kidney to be rendered more permeable, and there is evidence of this in the number of cases in which albuminuria occurs. McCrae put the incidence of albuminuria at 60 per cent. in a series of over 1,500 cases of typhoid (*System of Medicine*, Osler & McCrae), and in paratyphoid fever a trace of albumin is found in the urine quite commonly. Dieulafoy states that albuminuria does not occur until the second week, and disappears during the third week as a rule. Bacilluria is almost always, though perhaps not necessarily, associated with

albuminuria, but the frequent occurrence of albuminuria shows that there is a tendency for the permeability of the renal tissue to be increased. A similar association has been demonstrated in other diseases, notably in tuberculosis. Foulerton and Hillier, in cases investigated at the Middlesex Hospital, showed that out of six fatal cases of phthisis in which tubercle bacilli were found in the urine during life, albumin was also present in five. In these cases no trace of tuberculosis of the urinary tract could be discovered at post-mortem examination.

When bacilluria is present in the course of infectious fevers there may be no signs of inflammatory reaction in any part of the urinary tract, but in many cases there is some pyuria, though sometimes only amounting to the detection of a few pus cells in the urine. Still, pyuria is frequently to be found in the typhoid group of fevers if specially sought for, and may be taken as evidence of a mild pyelitis. The frequency of pyuria is difficult to ascertain with accuracy. A degree of pyuria which one medical officer would pass over as hardly worthy of mention, another would classify as a complication.

Review of Cases.—The cases of urinary disease included as complications in the present series comprise (Table IX), therefore, only those in which there were signs and symptoms directing attention definitely to some part of the urinary tract. The specific infecting organisms were present, in the

TABLE IX.—GENITO-URINARY COMPLICATIONS

Disease.	Cases.	Embol abscess.	Pyelitis.	Cystitis.	Prostatitis.	Epididymo-orchitis.	Totals.
Typhoid inoculated .	821	—	—	1	—	—	1
Typhoid not inoculated	297	1	2	1	—	—	4
Paratyphoid A inoculated	123	—	—	—	—	—	—
Paratyphoid A not inoculated . . .	221	—	1	—	—	—	1
Paratyphoid B inoculated	239	—	—	—	—	—	—
Paratyphoid B not inoculated . . .	799	—	6	5	1	4	16
Totals . . .	2,500	1	9	7	1	4	22

urine in all the cases. In one case of typhoid multiple embolic abscesses were found in the kidney at post-mortem examination. The patient died from general streptococcal infection, with endocarditis and, during the last few days, erysipelas. Pyelonephritis is a rare complication of the typhoid group of fevers, and perinephritic abscess, of which there was no instance in this series, has also been observed.

Occasionally the disease falls with particular violence on the kidneys at the onset, an acute nephritis masking for a time the true cause of the patient's illness. Amongst the cases of nephritis admitted to the wards reserved for cases of trench nephritis were a few patients who were found later to have typhoid or paratyphoid fever, but they were probably suffering from both con-

ditions, and the nephritis may have been independent of the typhoid group infection. There was only one case due to typhoid group infection which was of any severity. This occurred in paratyphoid B, and is described on page 119.

Pyelitis and cystitis were definitely diagnosed in sixteen cases, and as a rule there was distinct evidence of a descending infection of the urinary tract. First pyuria was observed, and this was followed later by frequency of micturition and other signs of infection of the bladder. The time of infection is generally about the third week or later, and of the sixteen cases, ten began after the third week. The renal pelvis may be infected either by the organisms which are being excreted by the kidney, or by those in the blood supplying the mucous membrane. In cases of cystitis also, the infection may be carried to the bladder directly by the blood stream, or the bladder may be infected by the urine. In the cases which I have been able to follow throughout their course, cystitis always followed an obvious pyelitis, and seemed to be due to a descending infection of the urinary tract. This was especially noticeable in three cases of paratyphoid B, which were complicated first by pyelitis, then by cystitis, and eventually developed epididymo-orchitis, with abscess in the testis from which bacillus paratyphosus B was isolated in pure culture.

Bacillus paratyphosus B proved more virulent in the urinary tract than the other organisms of the

group. There were not only more cases of infection due to the bacillus, but the cases were more acute and more extensive. Having a distinct bearing on this point is the fact that there was one case of prostatitis and urethritis, and there were four cases of epididymo-orchitis in paratyphoid B and none in the other two members of the group.

In one case of paratyphoid B the main stress of the illness fell on the urinary tract, starting with very acute pyelitis with some hæmaturia, followed by cystitis and eventually a suppurating orchitis. Pure cultures of bacillus paratyphosus B were obtained from the urine and from the pus out of the abscess of the testis. A similar case of acute urinary infection in paratyphoid B was reported in the *British Medical Journal* of May 6th, 1916, by Worster-Drought and Mills Kennedy.

Treatment.—Typhoid and paratyphoid pyelitis and cystitis as a rule yield readily to treatment with urinary antiseptics. The administration of urotropine, with the addition of acid sodium phosphate if the urine is neutral or its acidity is low, leads to rapid improvement. Castellani states that urotropine "is occasionally useful in preventing complications in the gall-bladder and urinary bladder," and on the first appearance of bacilluria, if not before, it is wise to give urotropine, lest any more active infection of the urinary tract take place.

In the present series no case required local surgical treatment. But sometimes infection of the urinary tract persists for some time after the patient gets

about, and some patients were sent to England still passing bacilli and pus in the urine. This point is referred to again in the chapter on the Carrier Problem (page 167).

Incontinence and Retention of Urine.—When patients are in a state of semi-stupor there may be incontinence of urine, and great care must be taken to keep the external genitals as dry and clean as possible, for septic conditions are very liable to develop and sloughing may take place, especially in women. When urine escapes involuntarily, the possibility of the symptom being due to distension with overflow must always be thought of.

Retention of urine is well known to occur in the course of these fevers, and it was noted in eight cases of the present series.

Prostatitis with and without abscess formation has been observed in the course of these fevers, sometimes in association with inflammation of other parts of the urinary tract. The specific micro-organisms have not been isolated from the pus of prostatic abscesses during life, but have been obtained in pure culture from the prostatic recesses, seminal vesicles, and urethral crypts at post-mortem examination when suppurative spermocystitis and prostatitis have been present.

There was one case of prostatitis in the present series, and it did not go on to abscess formation. The complication started on the 23rd day in a case of paratyphoid B, and cleared up in about three weeks. Bacillus paratyphosus B was obtained in

pure culture from the urine during the attack of prostatitis, but not at any other time, in spite of repeated examinations.

Urethritis has also been reported as a complication of these fevers, but the bacilli of the typhoid group have not been isolated from the urethral discharge. In the present series there was no case of urethritis except those which could be accounted for by venereal infection.

Late Effects of Infection of the Urinary Tract.— The part played by bacilluria in the etiology of surgical diseases of the kidney is a subject which requires further investigation. Excretion of micro-organisms by the kidneys takes place in a large number of infectious fevers of every degree and severity, from the most acute, like influenza, to the most chronic, like tuberculosis. The permeability of the kidney tissue is increased, and albuminuria is common.

Bacteriuria and slight pyuria are certainly commoner than has been generally taught, and these conditions frequently arise in diseases which in their mildest forms may easily be classed as more or less minor ailments. A history of previous illness assumes, therefore, an increased importance in the case of disease of the urinary tract.

It is important to bear this in mind when investigating cases of urinary disease either during the course of, or after the apparent recovery from, an infectious fever. Only by a careful bacteriological examination of the urine and of any parts of the

urinary tract which may be explored at operation, and also of any contents such as pus or calculi, can we get a true idea of the part played by these specific micro-organisms as an etiological factor in diseases of the urinary tract.

Bacterial excretion may have some relation to the *formation of calculi* in the kidney, for the epithelial débris and coagulable deposits resulting from one of these infections may provide a nucleus for the deposit of salts which would otherwise have remained in solution in the urine. It is of great importance to add to our information on this subject, and urinary calculi should be submitted to bacteriological as well as chemical investigation.

A case which has some bearing on this point is one reported by Davies and Walker Hall (*The Lancet*, November 8th, 1913), in which over five years after an attack of typhoid fever a woman, who was found to be excreting typhoid bacilli in her urine, had her right kidney explored and ten calculi removed. The urine from the right side contained much pus, and yielded a pure growth of bacillus typhosus. From the left side urine free from pus and bacilli was obtained. The contents of the renal cavity, obtained at operation, yielded a pure growth of bacillus typhosus, and this organism was isolated from the calculi also.

This case is certainly not unique, and others of a similar nature will be recognised if sought for and worked out in the same thorough manner. Even if

the specific organisms of an infecting fever are not found in cases of urinary disease which follow such an infection, the urinary condition may still be due to the specific infection, but the original causal bacteria have disappeared.

CHAPTER IX

THE GENITAL ORGANS

Occasionally during typhoid and paratyphoid fevers gangrene of the external genitals may occur, usually in the female. Abscess of Bartholin's glands has been observed and typhoid bacilli have been isolated from the pus. Abscess of the ovary and of the Fallopian tube have been described with bacillus typhosus as the infecting organism, while infection with typhoid bacilli of existing ovarian cysts (generally dermoids) has also been met with. In the present 2,500 cases, which includes only fourteen female patients, there were none of these complications.

Epididymo-Orchitis.— Epididymo-orchitis is an occasional complication, and there were four cases in the present series. All four occurred in paratyphoid B, and bacillus paratyphosus B was obtained in pure culture from three of them in which suppuration took place. The suppurating cases were inoculated with typhoid vaccine only, the one which did not suppurate had had a mixed vaccine (T.A.B.). The time of appearance of this complication was between the 30th and 40th days,

which corresponds with the usual time of onset in typhoid cases – late in the disease or in convalescence. There was some rise of temperature for a few days, but apart from this there was no general disturbance.

Pathology.—Owing to the body of the testis being involved as a rule either alone or to the same extent as the epididymis, it is generally taught that the infection in typhoid and paratyphoid fever is carried to the testis by the blood stream. But the organisms of these diseases give rise to so little infiltration that the infection may extend from the urinary tract to the seminiferous tubules before producing an obvious inflammatory reaction. It is different with organisms like the gonococcus, the pyogenic cocci, and the tubercle bacillus which cause considerable thickening and infiltration along the path of their extension, which leads to the epididymis being involved definitely before the testis in these infections. In the case of tuberculous infection, in which disease the infiltration and thickening are most marked, the reaction brings about a focus of inflammation first at the globus minor where it may be arrested and limited. In the four cases under review it certainly could not be said that the epididymis was first involved or even markedly affected, in fact the abscesses were all in the body of the testis. In each of the suppurating cases, however, there had been pyelitis and cystitis, and in the case which did not go on to suppuration there was very definite bacilluria, and bacillus

paratyphosus B was obtained in pure culture from the urine of all of them.

Taking these points into consideration, and bearing in mind that there was no great general disturbance with abscesses in other parts of the body, it seems reasonable to conclude that the infection in these cases came from the urinary tract, and I believe that many cases which are attributed to infection from the blood stream may be accounted for in the same way.

Mastitis.—The breast does not escape involvement during typhoid group infections, and in most large series of cases a few instances of mastitis with or without abscess formation will be found. It may occur in men, but naturally the bulk of the cases are in women, and in the present series there was no case of mastitis. The usual time for this complication to arise is about the third week during the stage of defervescence, less commonly in convalescence or after the patient is well. It is said that about half the cases go on to suppuration, and bacillus typhosus has been recovered from the pus in a few instances.

CHAPTER X

PAROTITIS

PAROTITIS is a serious complication, not only as an indication of the septic condition of the patient and of his mouth in particular, but also in itself as an inflammatory or suppurating focus, which is under high tension, because of the firm character of the fascia covering the gland. Inflammation may be so acute as to lead in rare instances to necrosis of the gland, or cranial and intracranial complications may arise, or general systemic infection develop. Of the nine cases in the present series, two were fatal, one of the patients suffering from typhoid and one from paratyphoid B. The typhoid patient was in a serious toxic condition, and died from pneumonia on the 17th day, parotitis having begun on the 12th day. The patient with paratyphoid B was severely ill from the first, and suffered from intestinal hæmorrhage and laryngitis, as well as parotitis, which appeared on the 22nd day, and death occurred on the 28th day of the disease. Intestinal hæmorrhage was a preceding complication in four of the nine cases, and by causing anæmia and some dryness of the mouth

may have been a contributing factor in favouring the parotid infection. The administration of morphia may also favour the development of parotitis. The usual time for parotitis to develop is in the third week or after, and in the present series only two cases occurred before the third week, five out of the nine beginning after the 30th day.

As a rule only one parotid is affected, and with one exception all the cases of the series were unilateral. Other salivary glands may also be affected, but there was no instance in this series. About half the cases suppurated—the usual proportion in other collections of cases. The gland was incised in six of the nine cases, but in two of them no pus was found—not an uncommon experience in secondary parotitis, for the symptoms and signs may be very deceptive. Still, having regard to the dangers of the condition, incision should be made, without waiting for fluctuation, when the swelling increases or œdema appears. The specific bacteria of the typhoid group were not found in any of the present cases, but bacillus typhosus has been found as the infecting organism either alone or with the pyogenic cocci; as a rule, however, cocci alone are found, and most frequently staphylococci.

Pathology.—Accumulating evidence goes to show that infection extends to the gland through the parotid duct. No doubt infection may reach the parotid gland by the blood stream as well as by Stenson's duct, but the great majority of cases,

if not all, can be explained satisfactorily by infection along the duct. The parotid may be the seat of an embolic abscess, but with such a manifestation of pyæmia suppurating foci will be present in other parts of the body, and parotitis arises extremely rarely in this way. In the present series there was no case of a pyæmic nature. Moreover, microscopic examination in fatal cases shows the inflammatory changes to be confined in the first place to the ducts, centres of the lobules, and the tissues around, the blood vessels being primarily unaffected. The subject of secondary parotitis was dealt with fully by Bucknall (*Med. Chir. Trans.*, 1905, lxxxviii. pp. 1–33; *The Lancet*, October 21st, 1905), who not only collated the evidence in favour of the duct infection theory, but gave a complete account of the whole subject and published a series of convincing cases. He came to the conclusion that all cases are due to an ascending infection of the parotid duct.

In typhoid and paratyphoid fevers, besides the lowered vitality of the patient and increased liability to septic infection, there are certain local conditions which predispose to an ascending infection of the duct. Owing to fever and the restricted diet, the salivary secretion is diminished in amount. At the same time the mouth breathing, so frequent in fever patients, leads to further drying of the mucous membrane, and micro-organisms become more plentiful in the mouth as well as more virulent. Moreover, the specific bacteria of the

disease have frequently been demonstrated in the oral secretions.

Prevention and Treatment.—Too much attention cannot therefore be given to the toilet of the mouth, not only for the sake of the patient's comfort, but also for the prevention of this serious complication. It is well also to prevent constipation as far as possible, to avoid the use of opium, and to give solid food as soon as it is safe to do so. The head should not be kept too low. When the condition arises and suppuration supervenes, early incision is called for, but suppuration may often be prevented and the inflammation led to resolve by careful cleaning of the mouth, the administration of a purgative if not otherwise contra-indicated, and the exhibition of sialogogues.

CHAPTER XI

THE RESPIRATORY SYSTEM

The Larynx

A MILD catarrh of the larynx is of frequent occurrence in the course of typhoid and paratyphoid fever and often passes without special notice, but occasionally ulceration occurs, and also perichondritis with exfoliation of the cartilages. Even this serious condition may be very insidious in its onset and course, and may only be discovered at post-mortem examination.

Pathology.—The time of onset of the severe cases is generally in the third week or often later, and several cases have arisen during convalescence. They may be veritable tragedies, sometimes bringing about the death of the patient in a few days, when he seems to be progressing steadily to complete cure. Ulceration may be of two types, either being due to coccal invasion of fissures and erosions or to true typhoid ulcers in the lymphoid tissue normally present in the laryngeal mucous membrane. From these ulcers typhoid bacilli have frequently been obtained. The typhoid ulcers are excavated, and are generally found on the posterior

wall of the larynx, over the arytenoid cartilages and the posterior plate of the cricoid, and also on the base of the epiglottis. There is some difference of opinion as to whether perichondritis is secondary to ulceration or whether it is the primary lesion in the majority of cases. Keen came to the conclusion that perichondritis is the primary lesion in severe laryngeal disease, but this view is not accepted by all, and there are many who think that ulceration extends to the cartilages, and thus leads to necrosis. It must be remembered that ulceration is more common than perichondritis, and that the cartilages being very poorly nourished, their vitality may easily be interfered with by ulceration.

Treatment.—Severe laryngeal complications are extremely serious, and a very careful watch must be kept on patients suffering from laryngitis. At the first sign of perichondritis or severe dyspnœa or stridor tracheotomy should be performed. Keen and Lüning showed very clearly the danger of delay in these cases, and insisted on early tracheotomy, with division of the cricoid ring if this cartilage was involved. Stenosis may result from ulceration and perichondritis, and there may be very great difficulty in dispensing with the tube. The stricture may be most intractable, and Lüning found that of sixty patients who recovered from perichondritis, forty-nine had to wear the tube permanently.

In the present series (Table X), no doubt many slight attacks of catarrh passed without being noted, but a definite laryngitis is described in

17 cases, 10 occurring in typhoid and 7 in paratyphoid B. The severer cases all developed after the third week.

TABLE X

Disease.	Cases.	Laryngitis.	Fatal.	Tracheotomy.	Fatal after Operation.
Typhoid inoculated . .	821	2	—	—	—
Typhoid not inoculated .	297	8	3	2	1
Paratyphoid A inoculated .	123	—	—	—	—
Paratyphoid A not inoculated .	221	—	—	—	—
Paratyphoid B inoculated .	239	1	—	—	—
Paratyphoid B not inoculated.	799	6	—	—	—
Totals	2,500	17	3	2	1

Three of the typhoid cases were fatal. One patient with extremely severe typhoid infection died of diffuse bronchitis, and œdema of the larynx was found on post-mortem examination. In another serious toxic case with broncho-pneumonia, ulcers were found at autopsy and the epiglottis, true cords and arytenoid cartilages were exposed. In the third fatal case tracheotomy was performed, but although the obstruction was relieved, death occurred three days later. This patient also had severe bronchitis and erysipelas. Some idea of his septic condition can be gained from the accompanying photograph (Fig. 24). At post-mortem examination it was found that the false cords were very swollen, while the surrounding parts were œdematous. The mucous membrane was

covered with sloughs, and the epiglottis was very thick and swollen. My friend Colonel Davies-Colley performed a timely tracheotomy in another case of ulceration of the epiglottis and ary-epiglottidean folds, and the patient recovered, but when he left France he still had to wear a tube. All attempts

Fig. 25. Patient who died of septicæmia with erysipelas and ulceration of the larynx.

at managing without it were unsuccessful. The other cases were much less severe, and improved with inhalations and medical remedies.

Paralysis of the laryngeal muscles, generally due to peripheral neuritis, is a condition which occasionally arises, and included in the table are two cases of paralysis of the vocal cords which occurred in paratyphoid B.

Lungs and Pleura

Other complications of the respiratory tract which may require surgical treatment are lung abscess and empyema.

TABLE XI

Disease.	Cases.	Lung Abscess.	Empyema.	Dry Pleurisy.	Pleurisy with effusion.
Typhoid inoculated . .	821	—	—	4	3
Typhoid not inoculated .	297	—	—		
Paratyphoid A inoculated .	123	—	—	3	1
Paratyphoid A not inoculated .	221	—	—		
Paratyphoid B inoculated .	239	—	—	9	6
Paratyphoid B not inoculated .	799	3	2		
Totals	2,500	3	2	16	10

Chest complications are comparatively common in typhoid and paratyphoid fever, but it is only rarely that surgical intervention is required. Lung abscess or empyema may arise, however, either from a primary pneumonia or pleurisy or from extension of suppurative foci in the abdomen, and the organisms of the typhoid group have been found in the pus taken from cases of empyema and abscess of the lung arising in either way.

In the present series there were three cases, and all occurred in paratyphoid B. In one case which proved fatal from broncho-pneumonia the lung was found at the post-mortem to have advanced in places to necrosis and abscess formation.

Bacillus paratyphosus B was isolated from the lung abscesses. It was obviously not a case for surgical intervention. The other two cases of lung abscess have been referred to in the chapter on complications involving the liver. One case, with appendicitis and portal pyæmia, developed empyema and abscess in the lower lobe of the lung from extension of a liver abscess through the diaphragm. The other also was not a true case of lung abscess, but one in which the pus from a liver abscess was expectorated after rupture into a bronchus. The second case of empyema was an interlobar collection which was drained with success thirty days after an unresolved pneumonia began. The organism present was the pneumococcus. The incidence of pleurisy is shown in the table, and the case of interlobar empyema was the only one which went on to suppuration. An interesting case of empyema due to infection by bacillus paratyphosus A has been reported by Capt. C. C. Weeks (*The Lancet*, September 2nd, 1916).

CHAPTER XII

JOINT COMPLICATIONS

THE joint affections met with in the course of typhoid and paratyphoid fever may be divided into :—

1. Acute arthritis, which may be (*a*) serous or (*b*) suppurative.
2. Subacute arthritis, which is of a serous type, but may also go on to suppuration.

1. (*a*) **Acute Serous Arthritis.**—Quite early in the disease, even at the onset, one, or more often, *many* joints may be the seat of an acute inflammatory effusion producing a condition very like acute rheumatic arthritis and sometimes mistaken for it. When the true diagnosis is established, it becomes evident that what had been taken for rheumatic fever was an arthritic manifestation of typhoid or paratyphoid infection during the stage when there are many bacilli in the general circulation, and all parts of the body are open to the attack of the specific bacteria.

The usual termination of an arthritis of this sort is for complete resolution rapidly to take place.

No special treatment is indicated, but a very careful watch must be kept on the joints, as occasionally suppuration may take place. A certain amount of stiffness may result in some cases unless movements are permitted as soon as possible.

TABLE XII

Disease.	Cases.	Acute Serous Arthritis.	Acute Suppurative Arthritis.	Subacute Arthritis.
Typhoid inoculated .	821	1	—	1 Hip (aged 19), onset 31st day
Typhoid not inoculated	297	—	—	— —
Paratyphoid A inoculated	123	—	—	— —
Paratyphoid A not inoculated	221	—	—	1 Knees (aged 30), onset 17th and 18th days
Paratyphoid B inoculated	239	—	—	1 Shoulder (aged 22), onset 18th day
Paratyphoid B not inoculated	799	2	—	1 Hip (aged 33), onset 24th day
Totals . . .	2,500	3	—	4 —

In the present series there were three instances of this type of joint affection, and one of the cases bore a close resemblance to rheumatic fever in the early symptoms, but the administration of salicylates had no effect on his condition. Abdominal discomfort and diarrhœa led to a suspicion of enteric group infection, and bacteriological investigation proved the case to be one of paratyphoid B. Another patient with paratyphoid B suffered from acute arthritis in many joints, beginning on the eighth day of his disease and subsiding in a few

days. The third was a case of typhoid fever complicated early in its course by pain and effusion in many joints which soon resolved. The fact that they occur early in the disease during the stage of marked bacillæmia points to these joint reactions being directly due to the specific bacteria of these fevers. Treatment is carried out on the usual lines.

(*b*) **Acute Suppurative Arthritis.**— Septic arthritis in typhoid or paratyphoid fever is a dangerous complication and usually proves fatal. It may follow an acute serous arthritis, or a subacute arthritis may become purulent, or again septic arthritis may be an incident in a general pyæmia or may be the result of extension of suppuration from an osteomyelitis in the neighbourhood of a joint. On the mode of origin of the septic arthritis will depend the time of its onset and also the number of joints affected. Unfortunately, being caused as a rule by a general septic invasion, more than one joint is usually involved. There are many portals of entry for other micro-organisms besides those of the particular fever from which the patient is suffering, for besides the intestinal ulcers, boils or bedsores are frequently present in cases of septic arthritis. The possibility of suppuration occurring with typhoid infection alone has been proved experimentally by Ellis (*Journ. of Infectious Diseases*, 1909, vi. 181), who showed that by injecting cultures of bacillus typhosus into joints serous, sero-purulent or purulent arthritis might result. There is also

abundant clinical evidence of the pyogenic properties of the typhoid group of organisms, and bacillus typhosus and paratyphosus B have been obtained in pure culture from cases of purulent arthritis. As a rule, however, suppuration in a joint is due to a mixed infection and the prognosis is correspondingly grave. The first time that paratyphoid bacilli were recognised was when Achard and Bensaude isolated them from the pus removed from the sterno-clavicular joint of a child. (*Bull. et mém de la Société de Méd. des Hôp.*, 1896.)

Symptoms and Treatment.—In this series there was no case of septic arthritis. The symptoms and treatment do not differ from those in septic arthritis from other causes; but, in view of our experience in aspirating a bone abscess due to infection with bacillus paratyphosus B, the proviso may be made that if a case is met with in which the patient's general condition is good and in which there is no infection with other micro-organisms than the bacilli of typhoid or paratyphoid, aspiration of the joint must be tried and repeated if necessary before more radical measures are undertaken. Vaccine treatment is also a useful adjunct.

2. Subacute Serous Arthritis.—The type of arthritis in which surgical interest is chiefly centred in typhoid and paratyphoid fever is the subacute form in which a serous effusion takes place quietly into one or occasionally more joints. In an overwhelming number of cases this type is *monarticular*. Though subacute serous arthritis may appear when

the patient is still acutely ill, this type generally develops when the acute stage of the illness is over, in the third week or even in convalescence. It affects the larger joints, such as the shoulder, elbow, or knee, but more especially the hip, and the joints of the lower extremities much more often than those of the upper.

This type of arthritis is not much if any commoner than the others we have discussed, but its special characters are more striking and therefore individual cases are more often recorded, with the result that collections of published cases give the impression that it is much the commonest form of joint affection met with in these fevers. Hospital records, however, show that its frequency is about equal to that of the other forms. The effusion is not usually accompanied by much pain or general disturbance, though occasionally both are severe.

As a rule resolution gradually takes place, the effusion is absorbed, and all the functions of the joint are completely restored. Occasionally, however, some stiffness results, though this can be prevented by early movements and massage. Suppuration is very rare, but may supervene. In its very mildness and in the insidious character of its onset, however, lie the chief dangers of this form of arthritis in a "typhoid" patient, who may be extremely apathetic and dull. If the arthritis be overlooked, as it may be very easily in the hip or shoulder where large muscles mask the effusion, and if no steps are

taken to put the limb in a suitable position or to relieve the distension of the joint, spontaneous dislocation is very likely to occur. Especially is this so in the case of the hip joint and particularly in children. In 84 cases of this type of arthritis collected by Keen, spontaneous dislocation occurred in 43 (40 times in the hip, twice in the shoulder, and once in the knee), and 32 out of 35 patients were under 20 years of age.

Pathology.—More and more evidence is accumulating to show that this form of arthritis is directly due to the specific bacteria. In many cases on aspirating the joint no bacteria are found in the fluid, but from time to time one or other of the bacilli of the typhoid group is isolated. We have only succeeded in isolating the bacillus from one of our cases; a pure culture of bacillus paratyphosus B was obtained from the fluid withdrawn from a case of arthritis of the hip —the only place from which the organism was isolated in this case, though it had already been definitely diagnosed as paratyphoid B by agglutination tests. A striking feature, however, was the character of the effusions which were examined by Capt. A. G. Ritchie. The films were exceedingly rich in cells, a fact which rather points to the effusions having been poured out to fight bacterial invasion of the joints, and since lymphocytes were numerous, the invasions were probably by bacilli rather than cocci. It is very likely that the bacilli are to be found in many more of the joint effusions

of typhoid and paratyphoid fever if the joint be aspirated early. Orloff's experimental results (*Centralbl. f. Bakteriol.*, 1890, 366) point the same way, for after injecting joints of rabbits and dogs with cultures of bacillus typhosus, he found a large number of cells in the effusions which resulted, and in the earlier days bacillus typhosus.

Dislocation.—The occurrence of dislocation is to be explained by the gradual distension of the joint in a patient who is profoundly exhausted and apathetic, and in some cases at least the limb does not assume that position which is most conducive to the maintenance of joint stability, a point to which I shall return when describing the cases observed in the present series.

Opportunities of examining morbid specimens are very rare, as the patients generally recover, but when we consider the clinical history of these cases, it is evident that there cannot be any great destruction of joint elements. The capsule being stretched, any injudicious movement or change of position may easily lead to dislocation of the joint.

Cases in the Present Series.—There were four instances of arthritis of this type amongst the 2,500 cases of typhoid and paratyphoid fever which form the basis of the present review. In two cases the right hip was involved, in a third the right shoulder, and in the fourth case both knees were affected. They all began in the third week or later; one in typhoid fever, one in paratyphoid A, and two in paratyphoid B. They

illustrate various points in the course of such cases. For example, the shoulder case in paratyphoid B tended to become very stiff after the effusion was absorbed. Massage and movements restored the joint to full usefulness in three weeks' time. The knee case in paratyphoid A affected both knees, and illustrates the point that this form of arthritis may affect more than one joint. The occurrence of three cases in the lower limb to one in the upper, and two of these three cases developing in the hip joint, is in keeping with the usual distribution. In the hip case in typhoid fever there was not much effusion. The limb was flexed, but there was no abduction or eversion. The application of extension in the slightly abducted position soon brought the limb straight, and a month later he was able to walk about. In the case of arthritis of the hip which occurred in paratyphoid B there was considerable effusion and the patient lay in a lethargic and apathetic condition. There was no flexion or abduction or eversion of the limb. The position of flexion, abduction, and eversion assumed in ordinary cases of effusion into the hip joint, and in the early stages of tuberculous disease of the hip, tends to keep the head of the femur in the acetabulum. It is evident, moreover, that if no abduction or eversion, especially abduction, takes place, an increasing effusion into the joint must tend to dislocate the head of the femur. The dislocation takes place on to the dorsum of the ilium, for the strong ligament on the front of the capsule resists

distension, while the weaker posterior parts stretch, and make room for the effusion.

Treatment.—Treatment consists of relieving the distension of the joint and putting the limb in good position. The quickest, simplest, and most useful way of giving relief is to aspirate the joint. When the effusion is recognised, the sooner the joint is aspirated the better, for not only may damage be prevented, but valuable information may be obtained. After aspiration the limb should be put in good position. In the case of the hip the limb should be slightly abducted, and if flexion has taken place, this may be diminished gradually by weight extension. The treatment of these cases is simple as a rule, for suppuration is extremely rare and it is not usual for any stiffness to result. The danger is that the complication may be overlooked and there have been cases in which dislocation was the first thing to be noticed. It is essential that the slightest complaint of a typhoid patient should receive careful consideration and examination, and these cases only furnish another illustration of the dangers of treating any symptom lightly. Especially does this apply in this connection in the case of children. Not only has the physician to pay particular attention to the slightest complaint, but he has by frequent examinations to be always on the watch, lest any complication like arthritis develop insidiously without the patient complaining of any symptoms at all.

CHAPTER XIII

THE MUSCULAR SYSTEM

DEGENERATION takes place in the muscles in the course of many infections, but it is of special importance in typhoid fever not only because of the frequency with which it occurs, but because it proceeds to such a degree as seriously to damage the muscle fibres.

In the present series of cases we have met with some of the results of degeneration in paratyphoid B, so presumably it takes place in paratyphoid as well as in typhoid fever, though probably not to the same extent. Various forms have been described, such as cloudy swelling, granular, and fatty degeneration, but the most important and commonest is the hyaline degeneration described by Zenker in 1864, in which the muscles affected develop a waxy appearance. The process is most marked about the third week, at which time or later it may be so advanced as to leave the muscle fibres with so little elasticity that they become extremely friable. Groups of fibres and even whole muscles may rupture almost spontaneously or as the result of the very slightest exertion.

Small or large hæmorrhages result and hæmatomata are formed in the muscle sheath. Almost any muscle may be involved, but the commonest are the rectus abdominis, adductors of the thigh, pectoralis minor, the diaphragm, the transversalis, and other muscles of the abdominal wall. The involvement of the diaphragm may have a serious effect on the patient by interfering with full respiratory movements. The abdominal muscles are sometimes submitted to considerable strain even with a patient lying in bed very ill, and the rectus abdominis has been known to rupture in typhoid fever when the patient has been raising himself in bed, in cases of extreme meteorism, and from the strain of coughing or defæcation. The accident may pass unnoticed or be the occasion of sudden acute pain. In itself it is not a really serious complication, but it usually occurs in severe attacks of the disease. The hæmorrhages very frequently go on to suppuration, but in some cases absorption takes place, and repair may be complete. Occasionally a hæmatoma in a muscle may become the starting-point of a fibrous tumour.

When the hæmatoma is of any size, it is better evacuated when fluid, while if suppuration occurs, incision is called for. It has happened that the abscess has burst into the abdominal cavity, so any large collection of fluid should be opened without delay. Bacillus typhosus has frequently been isolated from the fluid evacuated from these hæmorrhagic and purulent collections. In the

present series complications occurred in the muscles three times in the course of typhoid and three times in paratyphoid B. One of the typhoid cases had a hæmorrhage into the right rectus abdominis, and bacillus typhosus was isolated from the fluid evacuated in the eighth week. The other two were fatal cases; one of them developed a large hæmatoma in the gluteal muscles of the left side, and, in the other case, two broken-down collections of blood were found in the rectus abdominis at post-mortem examination. There is no record in these cases of bacteriological examination of the fluid. One patient with paratyphoid B developed inflammation in the hamstring muscles on the 40th day, while in another an abscess in the rectus abdominis was opened, and in the third a large abscess developed in the lumbar region which we thought had arisen in muscles, though the point, as sometimes happens, was not an easy one to determine. The pus obtained from the abscesses in these cases of paratyphoid B was sterile.

CHAPTER XIV

BONE COMPLICATIONS

The bacillus typhosus has been found in the bone-marrow in a large proportion of fatal cases in which it has been sought for, and this tissue probably plays an important part in the pathology of the disease.

As the result of typhoid infection various pathological conditions have been found in the bones—including osteitis, osteo-periostitis, periostitis, and osteo-myelitis. Of these the commonest clinical manifestation of bone disease is an osteo-periostitis, but probably an infection of the bone-marrow is the primary underlying cause in all cases.

Bone lesions are rare during the course of typhoid and paratyphoid fever, and may be classed as sequels rather than as complications of these diseases, for they occur in convalescence or some time afterwards, generally months and in some cases several years after the patient is apparently cured—in one case as long as seven years after convalescence (Buschke, *Fortschritte d. Med.*, 1894, 573). In McCrae's series of 1,500 cases of typhoid fever there was only one patient with bone complication during the course of the illness. In the present series, though pain

in the tibia was complained of in about 1 per cent. of all the cases, there were only five instances of obvious bone disease. Four of them occurred in typhoid, and one in paratyphoid B, and four of the five were in non-inoculated individuals.

TABLE XIII

Disease.	Cases.		Bone lesions.
Typhoid inoculated . .	821	1	20th day ; swelling over third rib—subsided
Typhoid not inoculated .	297	3	30th day ; left lower ribs—subsided 25th day ; swelling over rib—subsided 21st day ; periostitis of tibia—vaccine treatment—subsided
Paratyphoid A inoculated .	123	—	—
Paratyphoid A not inoculated	221	—	—
Paratyphoid B inoculated .	239	—	—
Paratyphoid B not inoculated	799	1	32nd day ; abscess in connection with ninth costal-chondral junction aspiration—subsided
Totals	2,500	5	—

Pathology and Course.—Bone complications occur more commonly in adults than in children, but very rapid growth has frequently been observed after typhoid fever in children which suggests that there is a hyperæmic condition of the bones near the epiphyseal lines. While any bone may be affected, the commonest ones are the tibia, the ribs, and the femur, ulna, and humerus. The large number of cases affecting the tibia makes the number in the

lower limbs greater than those of the rest of the body put together. In the present series there were four instances of the ribs being affected and one of the tibia, apart from those cases of pain in the tibia in which there was no objective sign of disease. Only one bone is affected as a rule, but there may be more than one involved, and in one bone there may be two or more lesions. As a rule a localised but sometimes a diffused swelling appears on some superficial bone and usually goes on to suppuration, but may settle down. An operation being undertaken, some underlying osteitis is practically always found. In other cases localised necrosis may take place and a sequestrum be formed. An osteomyelitis with central necrosis is also described, but necrosis is rare in typhoid osteitis compared with that due to other organisms. A form of abscess met with especially in typhoid osteitis of the tibia is what has been called the shirt-stud abscess (bouton-de-chemise), in which a collection of pus under the periosteum is continuous through a channel in the compact bone with another collection in the bone itself or in the bone-marrow.

If the node or swelling subside, it may recur or it may not; recurrence sometimes takes place more than once, and eventually the case may end in suppuration. The pus from these abscesses yields a pure culture of the specific organisms in a remarkable number of cases. The explanation of this would appear to be that these complications arise either in convalescence or when the patient is

apparently well; that is, when all portals of entry for fresh infections are probably closed. One is forced to the conclusion that in a certain number of cases of the typhoid group of fevers the infective organisms linger in the bones, and when some im-

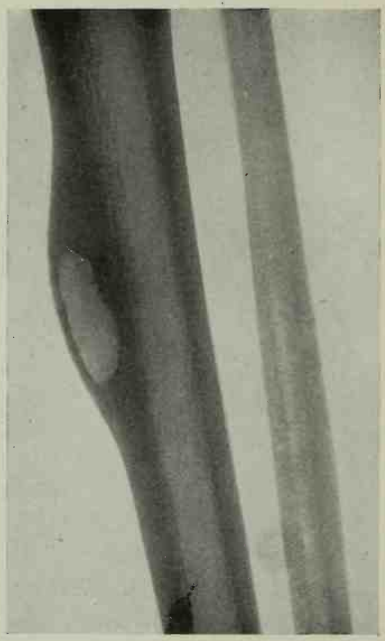


FIG. 26. Radiograph of typhoid osteo-periostitis of tibia (Knox).

pairment of general health or a local injury or strain provides a focus of lowered resistance, this is seized on by the typhoid or paratyphoid bacilli which have been lying latent in the bone-marrow all the time.

The general question of the persistence of typhoid group bacilli in the body will be discussed in the

chapter dealing with the Carrier Problem (see page 167). The bacteria still being in the marrow, the site at which infection will manifest itself may well be determined by local injury. Bones which are superficial and exposed to injury and strain are certainly those most commonly affected. In 216 cases collected by Keen, the tibia was affected 91 times to the fibula three times, and the ulna 15 times to the radius twice.

Secondary infection may occur or the bone disease may be caused by pyogenic cocci in the first place, in either of which cases the typical characteristics of the pure typhoid lesion will be lost, and severe damage may be done to the bone and the patient's life may be endangered. This is exceptional in a pure typhoid group infection. In fact the constitutional disturbance is usually very slight or there may even be none, and it is rare for any fever to occur. No doubt even in a pure typhoid group infection, serious or irreparable damage may be done to a limb, if the bone abscess extends into a neighbouring joint and even amputation may be necessary, but this is extremely rare.

Treatment.—It has been customary to advise radical interference by free excision of the obviously infected tissues, together with a good area surrounding, in those cases which do not subside spontaneously. It has frequently been found that if a partial operation has been done, a sinus has persisted for a long time, and in many cases a pure culture of bacillus typhosus has been obtained

from a sinus many months after the operation. In the ribs, wide resection has been found necessary before an area free from infection could be reached. No doubt if operation be performed in typhoid bone disease and the wound left open, the procedure must be radical and an attempt made to excise or sterilise all parts where the infection may be lurking; but I believe that if a sinus be avoided, less extensive measures may be successful.

In dealing with bone lesions we are only attacking a local manifestation of a lingering typhoid infection, and it is a big undertaking to attempt to rid the patient by operation of the whole infected area. Very extensive operations have been undertaken in cases in which the ribs have been infected, but I think other measures should be tried first. If treated by open operation, an attempt may be made to close the wound by suture in pure typhoid group infections. Armstrong (*Keen's Surgery*, vol. v.) has suggested puncture of the abscesses and the use of passive hyperæmia, but I think that in these cases we must have recourse to vaccine treatment in order to rid the body—not just this small bone area—of persisting typhoid or paratyphoid infection. Secondary infection with pyogenic cocci makes the case a much more acute and serious one, and it must then be dealt with by the free incision recognised as necessary in septic disease of bone.

Three of the five cases seen at the Isolation Hospital subsided spontaneously, and had not recurred when the patients left France (see Table XIII).

The case in which the tibia was affected in the course of typhoid fever did not subside at first, but after treatment with typhoid vaccine great improvement took place after an initial increase in symptoms during the negative phase. In the case which occurred in the course of paratyphoid B, a periostitis at the ninth costochondral junction went on to suppuration. From some pus withdrawn by syringe on the 32nd day of the disease a pure culture of bacillus paratyphosus B was obtained. On the 48th day some paratyphoid vaccine was injected with no effect. On the 55th day the abscess was aspirated, and a few days later there was nothing abnormal to be felt at the site of the lesion. A fortnight later the patient went to England, and there was still no sign of recurrence. These cases are very few, but the results are suggestive, and make one think that aspiration of typhoid and paratyphoid bone abscesses combined with vaccine treatment may make the more radical operations less often necessary. Many interesting cases successfully treated with vaccines have been reported recently by Emile-Weil (*Bull. de l'Acad. de Méd.*, 1917, No. 3).

Spondylitis

The term " typhoid spine " has been applied to a group of cases in which, during convalescence from typhoid or paratyphoid fever or some weeks afterwards, there is severe pain in the spine—generally

in the lumbar or dorsi-lumbar regions. The condition is rare, and like other bone lesions is a sequel rather than a complication of these fevers.

It may be regarded as a further instance of the special viability in bone of typhoid and paratyphoid bacilli. The lumbar and dorsi-lumbar regions are the parts of the spine most often affected, and Fraenkel (*Mitt. a. d. Greuzgebeit d. Med. u. Chir.* 1903, xii.) found typhoid bacilli more frequently in the lumbar vertebræ, probably because of the larger amount of bone-marrow in them.

The onset is generally within three months of convalescence, and may be acute or gradual. Symptoms may last for many months, but recovery is the rule. Pain, which is either local or referred or both, is the predominant symptom, and is aggravated by movement. It is associated with rigidity, dread of movement, with local tenderness, and occasionally some swelling. Spinal deformity develops in a fair proportion of cases. The condition follows cases of every degree of severity, generally occurs in the young, and is met with very much more frequently in men than in women. In some instances undue exertion or trauma during convalescence have apparently been exciting causes, and the part of the spine usually affected is certainly that most subject to strain, while the much greater frequency of its occurrence in male patients also suggests that muscular strain may have some influence in producing the condition. There is some rise of tem-

perature in nearly all cases. Signs of pressure on the cauda equina or the spinal cord are frequently present, and sensory disturbances, muscular paresis and atrophy, and changes in the reflexes have been observed. In some cases there are no very definite symptoms beyond the pain, and in the absence of objective signs it has been maintained that they are really due to functional disturbances—hysteria, neurasthenia, or neurosis, but this view cannot be upheld.

Typhoid spine was originally described by Gibney in 1889 (*Trans. Amer. Orthoped. Assoc.* 1889, ii. p. 19) as a "perispondylitis," by which he meant a periostitis with inflammation of neighbouring fibrous structures, and cases reported since X-ray examination has been the rule support the view that there is an organic lesion at the root of the trouble, and that this is generally a periostitis.

McCrae (*Amer. Journ. of Med. Sciences*, 1906, vol. cxxxii., p. 878) twelve years ago demonstrated the deposit of new bone in two cases, one after typhoid and the other after paratyphoid fever. Elkin and Halpenny (*British Journal of Surgery*, April 1914) have shown that in a large proportion of cases submitted to X-ray examination some organic change is revealed. Of 72 cases reported up to the end of 1908, only 11 had been examined by X-rays, but of 22 cases published between 1908 and 1914, X-ray examination was carried out in 14, and in 11 of them some lesion was demonstrated. The skiagrams show periostitis with effusion under the

periosteum, changes in the intervertebral discs and new formation of bone. Commonly only two adjacent vertebræ are involved, though rarely several may be. In rare instances with septic infection there may be suppuration, when caries and necrosis may occur; but in 94 cases of typhoid spine collected by Elkin and Halpenny, bare bone was stated to have been met with in two only.

Evidence of definite lesions in so many cases considerably reduces the number which might be attributed to neurosis, and even in many of these it is probable that there is some organic lesion underlying the condition. Those cases with symptoms due entirely to neurosis should not be classed as "typhoid spine." The clinical histories of the cases are so similar as to make it extremely unlikely that neurosis is a primary factor. Long-continued pain in a debilitated individual may well account for exaggeration of symptoms, but in the face of the frequent demonstration of skeletal lesions it is not reasonable to attribute the symptoms entirely to neurosis.

Prevention and Treatment.—The histories of published cases suggest that the development of this sequel of the typhoid group of fevers may be prevented in a certain number of cases by taking care during convalescence to avoid undue exertion. It also seems clear that if diagnosed and treated early, deformity and disability need not occur. The essential thing in treatment is rest, and when rest in bed has not given relief, weight extension or

fixation in an apparatus have been used with success. Recovery is very slow in some cases, but as a rule it is almost complete, though a little disability or rigidity or slight deformity may persist.

These sequelæ of typhoid and paratyphoid fever have an important relation to the Carrier Problem (see page 167), and have made us realise for many years that recovery from the disease does not mean death of all the infecting organisms. Keen insisted that a patient who had suffered from typhoid fever "should only be allowed to resume the ordinary activities of life very gradually, and should be cautioned as to even slight accidents and warned against any severe physical exertion for several months after he has apparently recovered."

CHAPTER XV

MISCELLANEOUS ABSCESSES. EYE AND EAR COMPLICATIONS

As pointed out in Chapter III, any organ, gland, or tissue of the body may be the seat of an inflammatory reaction as the direct or indirect result of typhoid or paratyphoid invasion, and abscesses may be found in any part of the body. Besides those which have already been described under various headings, cases have been reported of brain abscess and meningeal abscess without middle ear suppuration, as well as the numerous instances which occur in the connective tissues generally. Abscesses in the thyroid gland have been described, but are rare, and are more likely to arise in glands which were enlarged before the attack of typhoid. Bacillus typhosus has been recovered from them as a rule.

A review of the bacteriology of the cases of abscess in the present series is in agreement with the reports on other series of cases, and shows that, apart from organs and tissues in which the specific micro-organisms are constantly found, such as the spleen, liver, and the bone-marrow, it is the excep-

tion to obtain the bacilli of the typhoid group from foci of suppuration developing during the height of the disease. There is no question as to the pyogenic properties of the typhoid group of bacilli, for they have been proved time after time. A secondary infection may, however, outgrow the typhoid group organisms or the lesions may have been due originally to the pyogenic cocci or bacillus coli communis, which are the other bacteria most commonly found.

In a large number of cases a local cause can be found for the abscess, some lesion having provided a portal of entry for micro-organisms such as the pyogenic cocci, which have a greater chance of leading to suppuration in a patient who is already acutely ill and whose resisting powers are lowered. As examples of this we may take alveolar, axillary, perineal, or anal abscesses, or abscess in the buttock. These septic complications develop, as a rule, during the height of the disease. Complications which arise late in the disease, or in convalescence, or are met with as sequels, are generally due to the specific bacilli of the typhoid group, which in some cases may have been lying dormant for a long time.

In only one case out of twenty-two miscellaneous abscesses in the present series was the specific organism isolated, and that was from an anal abscess from which bacillus paratyphosus B was cultivated on the 19th day of paratyphoid fever. It must be remembered that pelvic and ischio-

rectal abscess may result from perforation of a typhoid ulcer of the rectum.

Boils were not common (there were only nine cases in which they were really troublesome), but they are said to be met with more frequently in patients who are having the bath treatment, a system which was not adopted at this hospital, and which could only be undertaken in exceptional circumstances at a War Base. Cases are recorded in which bacillus typhosus has been recovered from boils, but there were none in this series.

There were five cases of erysipelas, of which three were fatal. They all occurred in typhoid fever, and in non-inoculated individuals.

The Special Senses

The Eye.—With the exception of conjunctivitis, ocular complications of the typhoid group of fevers are rare, and there were none in the present series. The subject is essentially a special one, and has been dealt with from time to time by ophthalmic surgeons, notably by de Schweinitz (*Keen's Surgical Complications and Sequels of Typhoid Fever.* W. B. Saunders & Co., 1898).

As in other specific fevers, the ocular complications of typhoid and paratyphoid may be placed in three or four groups:

(1) Those associated with the febrile disturbance, such as conjunctivitis and superficial keratitis, which are of little importance.

(2) Those brought about by blood infection, which are the most important and also the most interesting of the eye complications. Iritis and cyclitis may be either of the suppurative type, when panophthalmitis may supervene, or of the plastic type, which may give rise to complete degeneration of the eye, detachment of the retina, cataract, etc. In this group de Grandmont's case (*Archives d'Ophthal.*, January 27th, 1892, p. 623) of iritis with hypopyon is of particular interest, for a pure culture of bacillus typhosus was obtained from the hypopyon, which was removed by paracentesis. Other deep infections cause choroiditis and optic neuritis, which may lead to optic atrophy. Orbital thrombosis and suppuration have also been described.

(3) Very rarely cases of paralysis of the extrinsic and intrinsic muscles of the eye have been reported.

(4) In the late stages of the disease, when the patient may be in an exhausted and semi-conscious condition, exposure phenomena, such as severe ulceration of the cornea with hypopyon, may develop from the eyes remaining open for long periods.

It is being recognised more and more by all surgeons, and by ophthalmic surgeons possibly more than others, that the infective fevers may be important etiological factors in certain obscure surgical conditions. Infections with various organisms which may be latent in some other part of the body may be responsible for many of the inflammatory conditions met with in the eye, or a persistent ulceration of a mucous membrane due to a previous

infection may provide a point of entry for organisms (see Charles Goulden: *Royal London Ophthalmic Hospital Reports*, vol. xix. Part III., November, 1914). This has always to be borne in mind when patients present themselves with inflammatory lesions of obscure origin for which there is no apparent cause. An inquiry into the patient's previous health should be very searching, and the absence of a clear history of an infective fever must not be accepted as negativing the possibility of such an infection having occurred. As already stated, infections with the typhoid group of bacilli may lead to only slight symptoms, and a history of some minor ailment may give a clue to the cause of an inflammatory condition. It is not only in the history, however, that evidence of a previous typhoid or paratyphoid infection may be found. The blood by its agglutination reactions may give proof of some previous or existing infection. A bacteriological examination of the fæces and urine, on more than one occasion if necessary, may throw light on an obscure inflammatory lesion of uncertain origin, for the patient may be a "carrier" of the bacteria of an infective fever.

The Ear.—It is comparatively common for some affection of the ear to arise during the course of these fevers. In the early stages of the fever patients frequently complain of buzzing in the ear, possibly owing to the low blood pressure. Deafness also occurs fairly frequently, coming on suddenly or gradually. There is no certainty as to its cause,

and though some cases may be due to neuritis, the majority are not, and it is exceptional for deafness to persist. The condition has been ascribed to changes in the labyrinth, such as hyperæmia, serous effusion, and hæmorrhages. In hyperæmia the prognosis is said to be good, as also with serous effusion, unless it be large in amount, but with a large effusion and with hæmorrhage the labyrinth may be permanently damaged.

It is with otitis media that the surgeon is specially concerned. This is the commonest ear complication and its frequency varies in different epidemics, occurring in from 1 to 7 per cent. of cases. Vergues (*Revue Hebdom. de Laryngol. d'Ot. et de Rhinol.*, September 28th, 1912) made a special study of the ear complications in an epidemic of 359 cases of typhoid fever. He divided the cases of otitis media into acute catarrhal and acute and chronic purulent. Acute catarrhal otitis media shows itself most often during the first ten days. It is not very painful, and often passes without being noticed. In the catarrhal type recovery is the rule, but it may change to an adhesive type or become purulent.

Purulent otitis media is commoner than the catarrhal form. It may be acute or chronic, and usually appears during the decline of the fever. The so-called acute type is generally rather subacute, and is therefore not associated with much pain. Otorrhea being present, it is more easily diagnosed than the catarrhal form. The patient may be convalescent when the temperature rises,

and in seeking for an explanation the medical attendant must not neglect to examine the ears. The perforation of the tympanum is generally single, and is in the usual situation—the antero-inferior quadrant. The prognosis is good, and recovery is complete in the greater number of cases, but the condition may become chronic. The mastoid antrum and cells are said by some to be involved fairly frequently, but this is by no means the usual experience. If a mastoiditis supervenes, any of the complications of this condition are also liable to occur. In the present series there was evidence of involvement of the mastoid in only one case, and that settled down without surgical intervention. As a rule, the pyogenic cocci only are obtained from the pus, but bacillus typhosus has been isolated in rare instances.

Treatment is carried out on the usual lines. The infection is conveyed from septic conditions in the throat and pharynx, and much may be done by careful cleansing of the mouth, nose, and throat to prevent the Eustachian tube from being infected and becoming a path of infection to the middle ear.

CHAPTER XVI

SURGICAL ASPECTS OF THE CARRIER PROBLEM

THE problem of the " carrier " of typhoid and paratyphoid bacilli is of great interest to the surgeon, and involves questions of considerable importance. The subject has been exhaustively dealt with by J. C. G. Ledingham and J. A. Arkwright in their book, *The Carrier Problem in Infectious Diseases* (Edward Arnold, 1912). In this chapter it is only proposed to deal with those points which seem to have some possible surgical significance.

The after-effects of this group of fevers have been discussed in the sections on the spleen, lesions of the bones, and of the biliary and urinary tracts. It has also been shown that chronic infection may be set up in the excretory channels by the organisms passing through them. In the biliary tract a chronic cholecystitis with or without gall-stones may result. In the urinary tract a chronic pyelitis or some other urinary complication may supervene, and, as in the case reported by Davies and Walker-Hall (*The Lancet*, November 8th, 1913), and already quoted on page 122, urinary calculi may be formed.

The carrier state is probably more likely to arise in those who have suffered during the course of the

disease from complications affecting these excretory channels—biliary and urinary—and it is therefore of the greatest importance to give careful attention to the treatment of such conditions and to keep under close observation those who have had symptoms of gall-bladder and urinary disease.

In a large number of "carriers" a lesion exists in one or other excretory channel. The "urinary carrier" generally has pyuria as well as bacilluria, showing that there is some inflammatory focus in the urinary tract. There may be seats of vegetation of the bacilli in the bladder, in the renal pelvis or in the various ducts and recesses opening into the urethra. The "intestinal carrier" in the great majority of cases appears to have some lesion in the gall-bladder, either with or without gall-stones. Forster (*Munch. med. Woch.*, 1908, p. 1) investigated a series of 173 chronic carriers, and found that 79 per cent. were women, a proportion which is very suggestive when it is remembered that the incidence of gall-stones is three times as frequent in women as in men. Moreover, although in only twenty-five cases, or 14 per cent., were there symptoms of cholecystitis, it is well known that the great majority of patients with gall-stones have no symptoms.

If typhoid or paratyphoid bacilli are being excreted in the urine or fæces, and a local lesion exists in the urinary tract or the gall-bladder, the local condition must be treated on ordinary lines. But, besides the cases in which lesions in the biliary or urinary tracts result from typhoid group infec-

tion, we have also to consider those cases in which bacilli are excreted either in the fæces or the urine for a long time after the patient is apparently cured, and in which there is no sign of disease in the excretory channels concerned. We may consider with this class also those cases which in spite of operative intervention, even with the removal of the part which has been the seat of an inflammatory condition, the "carrier state" persists. Before we can hope successfully to treat these cases, we must have a clear conception of the pathology of the condition, and must bear in mind that the biliary and urinary tracts are not the only parts of the body in which the bacilli can proliferate and lie dormant for long periods.

There remain then for consideration those cases in which radical operative intervention has not cured the carrier state, and those in which with the carrier state there is no apparent pathological condition in the urinary or biliary tract which requires surgical treatment.

Taking the *urinary tract* first, if bacilluria is associated with pyuria, it may be reasonable to regard a local condition in the urinary tract as one of the causes of the persistence of infection; but if there is no pyuria, the indication is to look elsewhere for some breeding-ground which may from time to time discharge bacilli into the blood stream, from which they are abstracted by the kidneys. Even with pyuria, and even if it is proved that excretion of bacteria is only taking place

through one kidney, it does not follow that the typhoid condition is purely a local renal one, and it is advisable also to bear in mind the possibility of some other infecting focus. The permeability of one kidney only may be increased, and a local lesion may only be facilitating the excretion of bacilli, and is not, necessarily, the breeding-ground of them.

In the case reported by Davies and Walker Hall (*The Lancet*, November 8th, 1913), with five years' history, bacilli were found in the blood at different times. The blood was examined during periods of bacillary excretion – four times it was found to be sterile, and twice yielded a strain of bacillus typhosus. It may be said that the bacilli were driven from the kidney into the circulation. Too much attention can be concentrated on the excretory channel, however, and the true explanation in some cases may well be that some other organ or tissue harbours the bacilli which find their way into the blood stream at intervals.

The bacilli may lurk in many places, but there are some which are known to be likely harbours. We know that in the case of typhoid fever the bacilli are to be found in the *bone-marrow* during the disease, and have been shown to be there as long as four months after recovery (Quincke, *Berlin Klin. Wochen*, 1894, No. 15) without causing symptoms. It is known that the bacilli may lie there, as other pathogenic bacteria are known to persist in the tissues in a latent state without

causing any disturbance. They are, however, always capable of doing so if the general health suffers or some local trauma reduces the powers of resistance at some particular point. Symptoms may then develop, and an abscess may result as long as over seven years after convalescence (Buschke, *Fortschritte d. Med.*, 1894, 573). This fact is very significant and suggestive when considered in relation to the carrier problem, for the bone-marrow is a tissue which is known to harbour the bacilli for long periods, and it is amongst the parts of the body most constantly infected. It has been shown repeatedly that the bone-marrow, the spleen, and the gall-bladder are the parts of the body in which these bacilli are most constantly found, both during the course of the disease and at long periods after convalescence. The bone-marrow must therefore be held under suspicion of being concerned as a harbour for the infecting organisms in those patients who do not rid themselves of infection when they recover from the disease.

Now let us consider *the biliary tract* and the persistence of typhoid or paratyphoid bacilli in the gall-bladder. Again attention has been concentrated on the excretory channel. It has been customary to look upon the gall-bladder, whether the seat of inflammation or not, as the great breeding-ground of typhoid bacilli when they have been found there at long periods after the original illness. No doubt there is a considerable accumulation of data pointing in this direction, but even if the gall-

bladder is the breeding-ground of the bacilli in the great majority of cases, there are some cases which do not conform to this conception of the condition. There are some in which no pathological condition has been found in the gall-bladder, and some in which the gall-bladder has been removed without success. There are other parts of the biliary and intestinal tracts which may be to blame, for example, the intestines or the smaller bile ducts or the liver itself. Keeping these factors in mind, it seems safer to regard the biliary tract as an excretory channel, and to look farther back for some other possible breeding-ground and source of continual or repeated infection. Many of the contents of the bile, including bacteria, are simply the sediment from the hepatic filter which has removed these substances from the blood coming to it from the gastro-intestinal tract and spleen, especially the spleen.

Knowing the important part *the spleen* plays in combating typhoid and other infections by abstracting bacteria from the blood, we may for the present purpose look on the bile as the splenic effluent. We know how active the spleen is in ridding the circulation of infecting organisms, sometimes enlarging to a great size to deal with the great strain thrown on it. This function of the spleen is probably seen at its best in those cases in which a cholecystitis is the sole manifestation of typhoid, and in those in which bacilli are discharged with the bile and fæces, and in which there have never been

any or only the mildest symptoms of typhoid or paratyphoid fever. But we also know that this organ may become a source of continuous danger, by harbouring infective organisms instead of ridding itself of them completely.

This is well known in certain protozoal diseases, and is being recognised to an increasing extent with regard to other infections. Other parts of the body may be cleared of infection by stimulating the natural resisting powers by means of vaccines or by administering chemical remedies, but the spleen still harbours the infective organisms. Splenectomy has been performed with success to rid the body of lingering infection in malaria (Jonnesco, *Bull. et Mem. Soc. de Chir. de Bucharest*, 1901–2, IV., 58) and in syphilitic and pyogenic infection (W. J. Mayo, *The Lancet*, November 25th, 1916). This organ is like a scavenger removing refuse, and thus protecting the community from many infective dangers, but if not scrupulously careful in attending to his own cleanliness, becoming a source of danger by harbouring infective material which ought to have been sent to the destructor. The dangerous characteristic of the spleen seems to have been recognised in the Elizabethan era, in the literature of which period we find many apt references to the spleen.

> "Though I am not splenetive or rash,
> Yet have I something in me dangerous."

And for a man to vent his spleen on a person was to give him of his worst.

In typhoid and paratyphoid fever we know that organisms are found most constantly in the spleen during the attack and in some cases for a considerable time afterwards. A man aged 21 died at the Isolation Hospital after being ill for 13 weeks and 5 days. Admitted for typhoid fever, which was proved by agglutination, he developed but few symptoms of typhoid and soon was found to be suffering from tuberculosis also. At the post-mortem examination he was shown to have died of miliary tuberculosis, but typhoid bacilli were still present in his spleen in large numbers, though there were no other signs of typhoid about him. In a case reported by Levy & Kayser in 1907 (*Arb. a. d. Kaiserl. Gesundh.* Bd. XXV., p. 254), a woman was found to be excreting typhoid bacilli with the fæces three years after having typhoid fever; she died, and typhoid bacilli were obtained from her gall-bladder and from inside a gall-stone, and also from the liver and spleen. Similar cases have been described, and are attributed to auto-infection, but some have died without symptoms of auto-infection, and yet typhoid bacilli have been found in the spleen.

The chain of evidence is not complete. Many tissues and organs may harbour the bacilli, but the spleen is the most likely, and is under the greatest suspicion. Surgery may not be able to help in ridding the body of infection if it is scattered through many organs and tissues, nor be able to extirpate all the diseased part if the infection is localised in the bone-marrow, but if localised in the spleen

and splenic effluent, surgical aid may yet be invoked and splenectomy be undertaken for persistent typhoid or paratyphoid infection.

When pathological conditions exist in the excretory channels, treatment will have to be directed to those parts, but the possibility of other breeding-grounds being responsible for the continuance of the infection must not be lost sight of, and when faced with the failure of other methods of treatment, operative intervention may be required to rid the body of an infective focus which is a danger to the individual and the community. The possibility of the spleen being the primary focus should be borne in mind, and when investigating the case of a patient who is a chronic carrier of bacilli of the typhoid group, useful information may be gained from blood obtained by splenic puncture, either without operation or when the abdomen is opened to deal with some surgical condition in the biliary tract.

INDEX

Abdomen,
  increased distension of, 68
  lung abscess and empyema arising from extension of suppurative foci in, 135
Abdominal pain,
  in intestinal perforation, 67
Abdominal tenderness, 68
Abdominal wall,
  degeneration of muscles of, 147
  large and tortuous veins developing in, 104
Abscesses, miscellaneous,
  bacteriology of, 160
  local causes, 161
  occurrence after convalescence, 171
  resulting from infection, 160
  see also Liver abscess; Lung abscess, etc.
Achard and Bensaude,
  first isolation of paratyphoid bacilli by, 42
Agglutination method of diagnosis, 49
Agglutinins,
  amount in patient's serum, how determined, 48
  observations on, 48
Albert, Prince Consort,
  death from typhoid fever, 25
Albuminuria,
  bacilluria frequently associated with, 116
  frequency in series of cases, 115
  time of appearance and disappearance of, 115
Alimentary tract,
  complications, 59–82
  portal of entry of the diseases, 50
Anæmia,
  following intestinal hæmorrhage, 61
Anæsthesia,
  in surgical treatment of intestinal perforation, 72
Anal abscess,
  isolation of p. bacilli from, 161
Appendicitis,
  and typhoid and paratyphoid, difficulty of differential diagnosis, 76, 77
  cases diagnosed as typhoid infection, 77
  complicating lung abscess, 136
  complicating venous thrombosis, 111
  diagnosis of, in cases of typhoid, 76, 77
  frequency of occurrence, 78
  non-typhoid, complicating typhoid and paratyphoid, 78
  pain of, compared with that of typhoid and paratyphoid, 77
  simulating typhoid and paratyphoid, 76, 77
  symptoms, 77, 78
Appendix,
  abscess complication, 79
  cases in which it should be examined, 80
  gangrenous, 79
    leading to hepatic abscess, 88
  involvement of lymphoid tissue, 77, 78
  perforation of, 63, 64, 65
    treatment, 73
  ulcer with large perforation (*illustration*), 77
  ulceration of lymphoid tissue of, 76
  usually primarily responsible for subphrenic abscess, 81
  with typhoid ulceration (*illustration*), 32
Army,
  first evidence of typhoid fever in, 19
  mild nature of paratyphoid A in, 53
Arterial thrombosis,
  characteristics, 103, 104
  following acute arteritis, gangrene due to, 103
  occurrence in both limbs, 106

Arthritis, acute purulent,
isolation of bacilli from cases of, 140
Arthritis, acute serous,
cases illustrating, 138, 139
diagnosis and symptoms, 138
frequency in series of cases, 138
rheumatic arthritis simulating, 137, 138
symptoms and treatment, 140
usual termination of, 137
Arthritis, acute suppurative,
causation, 139, 140
dangerous nature of, 139
occurrence with typhoid infection, 139
symptoms and treatment, 140
Arthritis, subacute serous,
character of the effusions, 140, 142
frequency of, 141, 143
frequent occurrence of spontaneous dislocation in, 142, 143
isolation of bacteria in, 142
joints chiefly affected by, 141
time of occurrence, 141
treatment, 141, 145
Auto-infection, 174

Bacillæmia,
of typhoid group infection, 47
Bacilluria,
B. paratyphosus B, 118, 119
frequency of, 115, 121
frequently associated with albuminuria, 115, 116
in relation to kidney disease, 121, 122
symptoms of, 116
time of appearance of, 115
treatment by urotropine, 119
Bacillus coli communis,
discovery of, 41
frequently in association with typhoid group bacilli, 161
in cases of acute cholecystitis, 93
isolation of, 51
Bacillus enteritidis of Gäertner,
discovery of, 41
Bacillus paratyphosus,
and food-poisoning bacilli, first distinction between, 42
discovery of, 42
influence of inoculation on, 57
isolation from cases diagnosed as typhoid, 42
portals of entry of, 50, 52
Bacillus paratyphosus (*continued*),
pyogenic cocci and bacillus coli communis frequently in association with, 161
pyogenic properties of, 161
tendency to suppuration, 54
Bacillus suipestifer,
discovery of, 41
Bacillus typhosus,
first demonstration in spleen and intestinal ulcers, 41
first isolation from the blood, 41
influence of inoculation on, 57
organisms distinguishable from, but producing identical symptoms and post-mortem appearances, 41
portals of entry of, 50, 52
pyogenic cocci and bacillus coli communis frequently in association with, 161
pyogenic properties of, 161
Bacteriological considerations, 51, 52, 54
Baillie, Matthew,
work on morbid anatomy, 35
(*portrait*), 34
Bartholin's glands,
bacilli isolated from pus of, 124
Bed-sores,
complicating septic arthritis, 139
thrombosis in relation to, 103
Bile,
constant presence of bacilli in, 92
infection carried by, leading to ulceration and perforation, 93
splenic effluent, 172
Biliary antisepsis, 95
Biliary tract,
bacilli present in, without development of gall-stones or cholecystitis, 102
bacilli present in, without signs of fever, 92
carrier state in relation to, 168, 171
cholecystitis occasionally only manifestation of infection of, 92
excretion by, 169, 172
infection of, predisposing to gall-stones, 101
symptoms, diagnosis and treatment, 92–6
inflammatory lesions in, 51
inflammatory reaction not obvious during hepatic excretion, 88

Biliary tract (*continued*),
local lesion in, 168
mode of entry of bacilli into, 102
pancreatitis dependent on inflammatory condition of, 102
presence of bacilli in normal condition of, 102
proliferation of bacilli in, 169
suppurative process leading to hepatic abscess, 88
Bladder,
prevention of complications by urotropine, 119
see also Cystitis
Blood,
activity of spleen in clearing infecting organisms from, 172
agglutination reactions affording proof of previous infection, 164
carrier state in relation to condition of, 170
changes in chemical composition of, 110
coagulability of, how increased, 61, 110
condition in case of hepatic abscess, 90, 91
condition in intestinal perforation, 68
condition in typhoid group infections, 49
eye complications due to infection of, 163
first cultivation of paratyphoid bacilli from, 42
first isolation of typhoid bacillus in, 41
infecting organisms separated by spleen, 51
influence of inoculation on bacteria in, 57
milk diet increasing calcium salts in, 110
percentage of cases in which bacilli are found in, 50
protective changes in, 51
Blood platelets,
causal relation to venous thrombosis, 110
increase of, 110
Blood vessels,
bacilli in thrombi and walls of, 109
impaired nutrition favouring occurrence of venous thrombosis, 109
Boils,
accompanying septic arthritis, 139
frequency of, 162
isolation of bacilli from, 162
Bone,
new, deposit in spondylitis, 157
Bone abscess, 151
isolation of organisms from, 151
Bone abscesses,
treatment, 155
Bone disease, 149–59
bones chiefly affected in, 150, 151, 153
causation, 152
formation of sequestrum, 151
frequency in series of inoculated and non-inoculated cases, 150
infection by organisms other than those of typhoid group, 153
isolation of bacilli, 155
local manifestation of lingering infection, 154
pathological conditions, 149
persistence of sinus, 153, 154
rapid growth in children, 150
secondary injection with pyogenic cocci, 153, 154
sequel rather than a complication, 149
swelling and suppuration, 151
time of occurrence, 149
treatment, 153–5
Bone marrow,
bacilli isolated from, 149
carrier state in relation to, 170, 171
special viability of bacilli in, 156
" Bouton-de-chemise " abscess, 151
Bowlby, Sir A.,
operation for intestinal perforation by, 71
Brain abscess,
resulting from infection, 160
Breast,
typhoid infection of, 126
Bronchitis, diffuse,
from typhoid cases of laryngitis, 133
Broncho-pneumonia,
complicating abscess of lung, 135
with typhoid laryngitis, 133
Bronchus,
rupture of pus from liver abscess into, 136

Cæcum,
paratyphoid ulceration in (*illustration*), 45

Cæcum (*continued*),
typhoid ulceration (*illustration*), 32, 33
Calcium salts,
avoidance in treatment of venous thrombosis, 112
coagulation increased by, 61, 110
Calf,
pain in, symptom of venous thrombosis of leg, 111
Cancrum oris,
fatal case of, 103
Cardio-vascular system complications, 103–114
Carrier state,
auto-infection in relation to, 174
biliary and urinary tracts chiefly responsible for, 167, 168
blood-stream in relation to, 170
bone-marrow in relation to, 170, 171
fæces and urine in relation to, 168, 169, 174
gall-bladder in relation to, 171, 172
harbours of infection after convalescence, 168, 170, 173, 174
"intestinal" carriers, 168
local lesions in relation to, 169, 170
nature of the complications favouring production of, 168
persistence after treatment and apparent cure, 169
production of, 51
protective inoculation in relation to, 58
spleen in relation to, 171–5
"urinary" carriers, 168
urinary tract in relation to, 169
Cauda equina,
signs of pressure on, 157
Cayley, Wm.,
(*portrait*), 27
Chest complications,
see Lungs; Pleura, etc.
Children,
rapid growth of bone complications in, 150
Cholecystectomy,
indications for, 95, 96
Cholecystitis,
age and sex incidence, 94, 98
bacilli in biliary tract giving rise to, 92
bacilli in biliary tract without presence of, 102
cases in which sole manifestation of infection, 172
Cholecystitis (*continued*),
cases without symptoms, 94
characteristics, 54, 93
conditions causing, 93
danger of perforation of gall-bladder in, 95
frequency in series of inoculated and non-inoculated cases, 98
gall-stones following, 100
gall-stones predisposing to, 95, 99
jaundice a prominent symptom of, 94
occurrence of, 54, 93
seldom associated with cholelithiasis, 94
symptoms and diagnosis, 93–5, 98, 99
symptoms in cases of gall-stones, 168
time of onset, 94
treatment, 95
see also Gall-bladder; Gall-stones
Choroiditis,
infection causing, 163
Circulation,
activity of spleen in driving infecting organisms from, 172
permanent damage to, from venous thrombosis, 104
sluggish, favouring occurrence of venous thrombosis, 109, 110
stimulation during convalescence, 112
see also Blood; Venous thrombosis, etc.
Citrates,
in treatment of venous thrombosis, 112, 113
predisposing to intestinal hæmorrhage, 112
Civil war,
typhoid fever during, 19
Collapse,
following intestinal hæmorrhage, 61
Colon,
perforation of, 63, 64
surgical measures, 72
with typhoid ulceration (*illustration*), 33
Complications,
characteristics and onset of, 51
general review of, 50–8
inflammatory products obtained from, 52
suppurative, 54

Complications (*continued*),
table indicating frequency and variety of, 53
typhoid more serious than paratyphoid in relation to, 53
Constipation,
complicating treatment, 106
prevention in venous thrombosis, 113
Continued fevers,
C. Murchison on, 27
differentiation of, by Sir Wm. Jenner, 25
progress in the study of, 29
Cornea,
ulceration with hypopyon, 163
Cyclitis,
suppurative and plastic, 163
Cystitis, 115
channels of infection, 118
characteristics and symptoms, 118–19
frequency of definite diagnosis in series of cases, 118
most active organism in, 118–9
time of onset, 118
treatment, 119

Dawson, Sir Bertrand,
clinical description of paratyphoid in present war, 46
Deafness,
causation, 165
frequent occurrence during infection, 164
Diabetes mellitus,
first recognised by Thomas Willis, 19
Diagnosis, 50, 51
importance of distinguishing between typhoid and paratyphoid, 53
serum, possibilities and employment of, 48
Diaphragm,
degeneration of, 147
Diarrhœa,
suggesting intestinal perforation, 69
Drainage,
intestinal, importance of, 73
Dysenteric ulceration,
and typhoid, differential diagnosis, 35

Ear complications,
during course of infection, 164-6

Eberth,
demonstration of typhoid bacillus in spleen and intestinal ulcers by, 41
Edward VII,
illness due to typhoid fever, 25
Embolic abscess,
in the kidney, case of, 117
Embolism,
gangrene resulting from, 103
Empyema, pleural,
frequency, 135
interlobar, 136
isolation of typhoid group bacilli from, 135, 136
Enterostomy,
indications for, in intestinal perforation, 73
Epididymo-orchitis,
cases illustrating, 118–9
isolation of bacilli from, 124
occasional occurrence of, 124
pathology of, 125
source and progress of infection in, 125
time of appearance, 124
Epiglottis,
ulceration of, 132, 134
Erysipelas,
complicating laryngeal ulceration, 133, 134
frequency of, 162
Escherich,
discovery of bacillus coli communis by, 41
Excretory channels,
carrier state in relation to, 167–74
Eye complications,
blood infection causing, 163
classification of, 162–3
complete degeneration in, 163
inflammatory conditions in, 163, 164
obscure, infective fevers important etiological factors in, 163
paralysis of muscles in, 163
rarity of, 162

Face,
gangrene about, 103
Fæces,
bacteriological examination in inflammatory lesions of uncertain origin, 164
excretion of bacilli by, long after apparent cure, 169
time of appearance of bacilli in, 50

Femoral vein,
fatal case of thrombosis of, 107
frequency of thrombosis in series of cases, 105
Fibula,
frequency of disease of, 153
Fraenkel,
isolation of typhoid bacillus from blood by, 41

Gäertner's bacillus, 41
Gaffky,
on typhoid bacillus, 41
Gall-bladder,
bacilli found at long periods after convalescence, 171
bacilli from pus and fluid in, 99, 100, 101
carrier state in relation to, 168, 172
cases in which it should be examined, 80
cause and mode of infection, 92, 93
channels of infection, 92
cholecystitis occasionally sole manifestation of infection of, 92
danger of perforation in cholecystitis, 95
empyema of, 93
harbouring bacilli at long period after convalescence, 174
infection of, symptoms, diagnosis and treatment, 92–6
local lesion in, 168
perforation of, characteristics and treatment, 96
prevention of complications by urotropine, 119
pure cultures obtained from, 92
Gall-stones,
bacilli in bile giving rise to, 92
bacteriological and chemical examination of, 122
cases predisposing to carrier state, 168
causal factor, 122
cholecystitis seldom associated with, 94
existence before infection facilitating development of cholecystitis, 95
experimental production of, 100
frequency in series of inoculated and non-inoculated cases, 98
history of infection in cases of, 101
Gall-stones (*continued*),
indications for removal of, 96
infection of, without symptoms, 94
infection through biliary tract predisposing to, 101
predisposing to paratyphoid cholecystitis, 99
rapid formation after onset of infection, cases illustrating, 100
relations of typhoid group infection to, 100
sex incidence, 168
symptoms observed to follow fever, 101
Gangrene,
about face and external genitals, 103
cause and treatment, 103, 104
of an extremity, cause of, 103, 104
of external genitals, 124
of the legs, 104
Gastric complications, 59
Genitals,
external, gangrene about, 103, 124
Geographical classification, 43
Glandular abscesses,
resulting from infection, 160
Gluteal muscles,
hæmatoma in, 148

Hæmatemesis,
in cases of typhoid, 59
Hæmatoma,
of muscle, treatment, 147
Hæmorrhage,
associated with intestinal perforation, relative frequency in series of cases, 70
muscular, 147
isolation of bacilli from, 147
splenic, 84
see also Intestinal hæmorrhage
Hamstring muscles,
inflammation of, 148
Heart,
abscess in wall of, 103
feeble action favouring occurrence of venous thrombosis, 109
Henry, Prince of Wales,
death from typhoid fever, 19, 20 (*portrait*), 20
Hip joint,
spontaneous dislocation in subacute serous arthritis, 142

Hip joint (*continued*),
subacute serous arthritis of, 142
dislocation of head of femur in, 144
frequency of, 143
treatment, 144, 145
Historical sketch, 19–42
Hunter, John, 29
description of cases of typhoid by, 31
on inflammation of peritoneum, 71
on perforation of gall-bladder, 96
on phlebitis and thrombosis, 108, 109
(*portrait*), 28
Hyperæmia,
in treatment of bone lesions, 154
of the ear, 165
Hypopyon,
isolation of bacilli from, 163
ulceration of cornea with, 163

Ileo-cæcal perforation, 65
frequency of, 63, 64
Iliac vein,
external, enlargement of superficial veins to compensate for obstruction of, 108
obstruction of, 104
fatal case of thrombosis of, 107
sudden onset of thrombosis of, 111
thrombosis of, 69, 105
(*illustration*), 108
Ileum,
exhibiting advanced stage of typhoid ulceration (*illustration*), 32, 33
inflammation of lymphoid tissue in, 51
obstruction by band above cæcum, 81
paratyphoid ulceration in, 44–5
perforation of, 65
perforation of large intestine more serious than that of, 74
Peyer's patch in (*illustration*), 44
showing Peyer's patches, with hyperplasia and early necrosis, 33
typhoid ulcer in, protected by peritoneal adhesion (*illustration*), 66
with enlargement of Peyer's patches (*illustration*), 30
Incontinence of urine, 120
Inflammatory products,
obtained from complications, 52
type of organism yielded by, 52
Inflammatory reactions,
occurrence of, 50
resulting from infection, 160
Inoculation,
effects on the course of the diseases, 54, 55, 58
frequency of carriers diminished by, 58
influence on bacteria, 57
intestinal hæmorrhage in relation to, 60
mortality rate, 55
statistical results, 54–5
Intestinal carriers,
observations on, 168
Intestinal hæmorrhage,
association with perforation, 70
citrates predisposing to, 112
essential factor in, 112
etiological relationship to complications, 52
gall-stones in fatal case of, 101
in case of typhoid parotitis, 127
mortality rate in cases of, 60
percentage in inoculated and non-inoculated cases of typhoid, 60
time of onset, 112
treatment, 60, 61
Intestinal lesions,
in paratyphoid A, 53
of typhoid and paratyphoid cases (*illustration*), 44–5
Intestinal obstruction,
conditions leading to, 81
following typhoid and paratyphoid, 67
in non-inoculated typhoid cases, 81
resulting from typhoid lesions in intestinal tract, 81, 82
Intestinal perforation,
age and sex incidence, 61
anæsthesia during operation for, 72
cases of peritonitis without, 81
characteristics, 64
commonest site for, 63
complicating venous thrombosis, 111
diagnosis, 68–70
false symptoms of, 69
frequency of, 62
hæmorrhage associated with, relative frequency in series of cases, 69, 70

Intestinal perforation (*continued*),
indications for laparotomy, 69
localisation of infection, 65
more frequent in paratyphoid than in true typhoid, 64
multiple perforations, 65
nature of the pain in, 67
pathology of, 64
percentage in inoculated and non-inoculated cases, 62
plastic peritonitis and adhesion of omentum in case of, 66
preventing or limiting infection of peritoneum, 66
prognosis, 73, 74, 75
relative frequency and severity in typhoid and paratyphoid cases, 74, 75
symptoms, 67
time of onset, 63, 74–5
treatment, 71–4
where lesions of true typhoid are most marked, 64
Intestinal ulcers, 30–3
first demonstration of typhoid bacillus in, 41
frequently present in septic arthritis, 139
healing of, 82
of peritoneal coat without inflammation of peritoneal surface, 80
Intestines,
abscess formation from escape of contents of, 65, 66
drainage of, 73
extent to which involved, 54
gangrenous inflammation leading to hepatic abscess, 88
harbouring bacilli after convalescence, 172
inflammation of lymphoid tissue in, 51
stricture following typhoid fever, 82
Iritis,
suppurative and plastic, 163
with hypopyon, 163

Jaundice,
as symptom of acute cholecystitis, 94
frequency in series of inoculated and non-inoculated cases, 98
frequency in typhoid and paratyphoid infection, 94
Jejunum,
perforation of, 63
Jenner, Edward,
statue of (*illustration*), 56
Jenner, Sir Wm.,
causation of typhus and typhoid differentiated by, 25
(*portrait*), 24
Joint aspiration, 140, 145
Joint complications, *see* Arthritis
Keen, W. W.,
on surgical complications and sequels of typhoid fever, 41
(*portrait*), 40
Kidneys,
bacilluria in relation to disease of, 122
cases illustrating involvement of, 117
causal relation of bacilluria to surgical diseases of, 121
embolic abscesses in, case of, 117
excretion of micro-organisms by, 121
permeability of, 115, 116, 121, 170
see also Pyelitis; Nephritis, etc.
Knee joint,
spontaneous dislocation in subacute serous arthritis, 142
subacute serous arthritis of, frequency of, 143, 144

Laparotomy,
indications for, 69
Laryngeal catarrh,
frequent occurrence of, 131
Laryngeal muscles,
paralysis due to peripheral neuritis, 134
Laryngeal stenosis,
cause, 132
Laryngeal ulceration, 131
broncho-pneumonia complicating, 133
causation, 131
dangerous nature of, 131
perichondritis less common than, 132
time of onset, 131
with septicæmia and erysipelas, death from (*illustration*), 134
Laryngitis,
frequency in series of inoculated and non-inoculated cases, 132, 133
mortality from, 133
serious nature of, 132
Legs,
gangrene of, 104
thrombosis in the veins of, 105

Leucocytosis,
degree met with, 49
Leucopenia,
production of, 49
Limbs,
persistent swelling resulting from venous thrombosis, 107
Liver,
changes in dullness of, 68
elimination of micro-organisms by, 88
exploratory puncture of, technique, 89
infection by hepatic artery, 88
mode of entrance of bacilli into, 102
multiple abscesses complicating typhoid appendicitis, 79
organisms not all killed by, 92
parenchymatous degeneration of, 51, 88, 92
sub-phrenic abscess behind, 81
thrombosis of veins of, 88
usual routes of infection of, 88
Liver abscess,
blood picture in, 90, 91
case illustrating symptoms, 90
cause and development of, 88
character of the pus from, 90
drainage of, 89, 90
exploratory puncture for, technique, 89
frequency of spontaneous rupture of, 91
isolation of bacilli from, 90
lung abscess from extension of, 136
pathology, 88
rupture of pus into a bronchus, 136
single and multiple, 88, 89
symptoms, diagnosis, and treatment, 89–91
Lung abscess,
causation, 135
frequency in series of inoculated and non-inoculated cases, 135
isolation of typhoid group bacilli from, 135, 136
Lymphocytes,
increase in number of, 49
Lymphoid follicles,
inflammation of, 63

Mastitis,
characteristics and time of occurrence, 126
Mastoid antrum,
involvement of, 166
Mayerne, Sir Theodore de,
illness of Henry, Prince of Wales, described by, 23
(*portrait*), 22
Meat poisoning,
discovery of bacillus of, 41
Meckel's diverticulum,
perforation of, 63, 73
Meningeal abscess,
resulting from infection, 160
Mesenteric glands,
cases in which to be searched for, 80
inflammation of lymphoid tissue in, 51
suppurating, peritonitis arising from, 79, 80
Micturition,
frequent, 118
Milk diet,
calcium salts in blood increased by injudicious persistence in, 110
Moore, Norman,
on the illness of Henry, Prince of Wales, 23
Morbid anatomy,
Matthew Baillie's work on, 35
Morphia,
favouring development of parotitis, 128
Mouth,
infection of. *See* Parotitis
Murchison, Charles,
on the continued fevers, 27
(*portrait*), 26
Muscle,
hæmatoma of, isolation of bacilli from, 147, 148
treatment, 147
hæmorrhage from, 147
hyaline degeneration, characteristics and appearance, 146
time of occurrence, 146
rupture of fibres of, 146
Myalgia and debility,
case of liver abscess reported as, 90

Nephritis,
complicating infection, 117–18
rarely due to typhoid infection, 118
Neuritis,
as cause of deafness, 165

Neurosis,
cases of typhoid spine with symptoms due to, 158

Ocular complications, *see* Eye complications
Œdema,
due to venous thrombosis, 104, 111
Œsophagus,
ulceration in typhoid, cause, symptoms, and treatment, 59
Omentum,
adhesion of, with plastic peritonitis in case of intestinal perforation, 67
Optic neuritis,
infection causing, 163
Oral secretions,
isolation of bacteria from, 130
Orchitis,
suppurating, 119
Osteo-myelitis,
acute suppurative arthritis from suppuration of, 139
with central necrosis, 151
Osteo-periostitis,
commonest clinical manifestation of bone disease, 149
of tibia (*illustration*), 152
Otitis media,
frequency, 165
purulent more common than catarrhal form, 165
time of onset, 165
Otorrhœa, 165
Ovarian cysts,
typhoid infection of, 124
Ovary,
typhoid infection of, 124
Oxford,
typhoid fever at, during the Civil War, 19
Oxygen administration,
in treatment of venous thrombosis, 113

Paget, Sir James,
on complications and sequelæ of the continued fevers, 39
(*portrait*), 38
Pain,
abdominal, in intestinal typhoid ulceration, 67
complications to which due, 67
Pancreatitis,
as complication and sequel, 102
cause and characteristics, 102
treatment, 102
Paratyphoid,
A and B, characteristics compared, 43, 44
and typhoid, clinical and post-mortem appearances compared, 43, 46
clinical description of, in present war, 46
complications of milder character than those of typhoid, 53
difficulties in detecting infecting bacilli in, 47
effects of inoculation on the course of, 54, 55
geographical classification, 43
mild character of, 53
mortality in series of inoculated and non-inoculated cases, 47
secondary infections outgrowing, 161
serum diagnosis of, 48
ulceration (*illustration*), 44–5
Parenchymatous changes, 51
Parotid duct,
channel of infection in parotitis, 128, 129
conditions predisposing to ascending infection of, 129
embolic abscess of, 129
necrosis of, 127
Parotitis,
cases illustrating condition of patient, 127
causal factors, 128
conditions arising from, 127
infecting organisms, 128
intestinal hæmorrhage as a preceding complication, 127
pathology, 128
prevention and treatment, 128, 130
secondary, 129
serious nature of, 127
time of occurrence, 128
Pelvic abscess,
resulting from perforation of typhoid ulcer of rectum, 162
Pelvic colon,
frequency of perforation of, 63, 64
Pericarditis,
purulent, 103
Perichondritis,
occurrence of, 131
stricture frequently resulting from, 132

Perichondritis (*continued*),
tracheotomy for, 132
ulceration more common than, 132
Perinephritic abscess,
rare complication, 117
Periostitis,
with effusion, characteristic in spondylitis, 158
Perispondylitis, 157
Peritoneal adhesion,
typhoid ulcer in ileum protected by (*illustration*), 66
Peritoneum,
intestinal perforation causing general infection of, 65
intestinal perforation preventing or limiting infection of, 66
Peritonitis,
cases of obscure causation, 80
cases without obvious perforation, 81
causes and conditions associated with, 79, 80
due to pyogenic organisms, severity of, 74
in fatal case of paratyphoid, 80
Peyer's patches,
portion of ileum showing (*illustration*), 30, 40
with hyperplasia and early necrosis, ileum showing (*illustration*), 33
Phlebitis, 108
preceding thrombosis, 109
symptoms, 111
symptoms of, before establishment of thrombosis, 113
Pleura, *see* Empyema
Pleurisy,
dry, frequency in series of inoculated and non-inoculated cases, 135
left-sided, indicating splenic abscess, 85
with effusion, frequency in series of inoculated and non-inoculated cases, 135
Pneumonia,
lung abscess and empyema arising from, 135
Polymorphonuclear leucocytes,
increase in number of, 49
Popliteal vein,
thrombosis of, frequency, 105
Portal pyæmia,
complicating lung abscess, 136
Portal vein,
hepatic infection through, 88
Prostatic abscesses,
isolation of micro-organisms from, 120
Prostatitis,
as complication, 119
characteristics and frequency, 120
time of onset, 120
with and without abscess formation, 120
Protean nature of the fevers, 50
Pulse rate,
in intestinal typhoid ulceration, 68
Pyelitis,
B. paratyphosus B more active than other organisms of the group, 118–19
cases of paratyphoid infection with, characteristics and symptoms, 118–19
cause, 118
channel of infection, 118
frequency of, 114
frequency of definite diagnosis in series of cases, 118
pathology, 114
pyuria a sign of, 116
time of onset, 118
treatment, 119
urotropine administration in, 119
Pyelonephritis,
rare complication, 117
Pyogenic cocci,
infection by, outgrowing typhoid group organisms, 161
inflammatory products yielding, 52
peritonitis due to, more dangerous than that due to typhoid bacteria, 74
secondary infection of bone lesions by, 153, 154
specific organisms of typhoid group in pure culture with, 51
ulceration of œsophagus by, 59
Pyogenic properties of typhoid group of bacilli, 161
Pyuria,
associated with bacilluria, 116
carrier state in relation to, 169
frequency of, 116, 121
symptom of mild pyelitis, 116

Radius,
frequency of disease of, 153
Rectal examination,
tenderness on, 68

Rectum,
perforation of, 63
typhoid ulcer of, pelvic and ischio-rectal abscess resulting from, 161, 162
venous thrombosis in relation to, 106
Rectus abdominis,
abscess in, 148
degeneration of, 147
hæmorrhage into, 148
rupture of, 147
tenderness and rigidity of, 68
Renal calculi,
bacterial excretion in relation to formation of, 122
case illustrating, 122
isolation of bacilli from, 122
Respiratory movements,
degeneration of diaphragm affecting, 147
Respiratory system,
complications and diseases of, 131–6
Retention of urine, 120
Rheumatic fever,
arthritic manifestation of typhoid or paratyphoid infection of joints mistaken for, 137, 138
Ribs,
disease of, frequency, 151
treatment, 154
splenic abscess pointing to œdema and redness over, 85, 86
Royal College of Surgeons,
John Hunter's specimens of typhoid ulceration in, 30–3

Salivary gland infection, *see* Parotitis
Saphenous vein, internal,
frequency of thrombosis in, 105
Sequestrum,
formation of, 151
Serum diagnosis,
difficulties of, 48
possibilities and employment of, 48
" Shirt-stud abscess," 151
Shoulder-joint,
spontaneous dislocation in subacute serous arthritis, 142
subacute serous arthritis of, frequency and treatment, 143, 144
Sigmoid flexure,
perforation of, 75
splenic abscesses in fatal cases of, 84
Simmonds,
isolation of typhoid bacillus from blood by, 41
Sodium citrate,
intravenous administration in venous thrombosis, 113
Spermocystitis, suppurative, 120
Spinal caries,
rare occurrence of, 158
Spinal cord,
signs of pressure on, 157
Spinal deformity,
development of, 156
Spine, " typhoid," *see* Spondylitis
Spleen,
activity in ridding the circulation of infecting organisms, 173
as primary focus of infection, 175
bacilli found in, at long periods after convalescence, 171
dangerous characteristic of, 173
enlargement of, function in course of the fever, 51
first demonstration of typhoid bacillus in, 41
function of, 173
hæmorrhage from, 84
infecting organisms harboured by, 171, 173, 174
isolation of bacilli from, 84, 174
multiplication of bacilli in, 102
pathological significance in typhoid group, 83
percentage of typhoid and paratyphoid cases in which palpable, 83
rapid enlargement as indication of abscess, 85
rupture of, 84
significance in typhoid and paratyphoid fevers, 87
suppurating infarcts in, 84
surgical conditions uncommon and difficult to diagnose, 83
Splenectomy,
dangerous nature of, 84
for splenic abscess, 86
indications for, 173, 175
Splenic abscess,
characteristics of cases of, 84, 85
diagnosis, 85
exploratory puncture for, technique, 86
frequency in series of cases, 84
in fatal cases of perforation of sigmoid flexure, 84
incision and drainage of, 86
infarction chiefly the cause of, 84

Splenic abscess (*continued*),
symptoms, 84, 85
treatment, 86
Spondylitis, 155–9
cases with symptoms due to neurosis, 158
causation, 156, 158
characteristics and rarity, 156
nature of the condition, 157
new bone deposit in, 157
organic bone change in, 157
parts chiefly affected in, 156
periostitis with effusion in, 158
prevention and treatment, 158, 159
symptoms, 156, 158
time of onset, 156
Staphylococci,
inflammatory products yielding, 52
Stenson's duct, *see* Parotid duct
Sterno-clavicular joint,
first isolation of paratyphoid bacilli from suppurative arthritis of, 42
Stomach,
ulceration in cases of typhoid, 59
Streptococcal infection as complication, 117
Subphrenic abscess,
appendix usually primarily responsible for, 81
Suppurative complications, 54
Symptoms, 50, 51, 54

Temperature,
changes in, 68
Testis,
source of infection of, 125
Thrombosis, *see* Arterial thrombosis; Venous thrombosis
Thyroid gland,
abscess resulting from infection, 160
Tibia,
abscess during osteitis of, 151
disease of, case illustrating treatment, 155
frequency, 150, 151, 153
osteo-periostitis of (*illustration*), 152
Tonsillitis,
etiological relationship to complications, 52
Tracheotomy,
indications for, 132, 134
Tuberculosis,
frequent association of bacilluria with albuminuria in, 116
Tympanites,
presence suggesting intestinal perforation, 69
Tympanum,
perforation of, 166
Typhoid,
advances in the study of, 41
and paratyphoid, clinical and post-mortem appearances compared, 43, 46
and typhus, differentiated by Sir Wm. Jenner, 25
differential diagnosis from paratyphoid, importance of, 53
difficulties in detection of infecting bacilli, 47
effects of inoculation on the course of, 54–5
historical sketch, 19–42
isolation of paratyphoid bacilli from cases diagnosed as, 42
mortality in series of inoculated and non-inoculated cases, 47
most serious of the group infections, 53
onset and course of, 50, 51
paratyphoid milder than, 43
protean character of, 50
resumption of ordinary activities of life following, 159
secondary infections outgrowing, 161
serum diagnosis of, 48
Typhoid group infection,
importance of detecting infecting organism, 46, 51
Typhus,
and typhoid, differentiated by Sir Wm. Jenner, 25

Ulceration,
Hunter's specimens (*illustration*), 30–3
ileum, cæcum, and colon with (*illustration*), 32, 33
Ulna,
disease of, frequency, 153
Urethritis,
as complication, 119, 121
Urinary antisepsis, 95
in pyelitis and cystitis, 119
Urinary calculi,
bacteriological and chemical examination of, 122
" Urinary " carriers,
observations on, 168
Urinary disease,
factor in diagnosis of, 122

Urinary disease (*continued*),
history of previous illness in relation to, 121
review of cases of, 116–19
Urinary tract,
B. paratyphosus B more active than other organisms in, 118–19
case illustrating paratyphoid B infection, 119
chiefly responsible for the carrier state, 168
descending infection of, 118
late effects of infection of, 121
local condition as cause of the carrier state, 169
local lesion in, 168
predilection of B. paratyphosus B for, 118–19
proliferation of bacilli in, 169
relative degree of irritation to, 54
Urine,
bacteriological examination in inflammatory lesions of uncertain origin, 164
bile in, in hepatic abscess, 91
excretion of bacilli by, long after apparent cure, 169
first isolation of paratyphoid bacilli from, 42
incontinence and retention of, 120
time of appearance of bacilli in, 50
see also Bacilluria
Urotropine,
advantageous use during course of infection, 95
biliary and urinary antiseptic, 95
in treatment of pyelitis and cystitis, 119

Vaccine treatment,
of typhoid bone lesions, 154
Varicose veins, 108
developing from venous thrombosis, 104, 107
Venous thrombosis,
calcium salt content increasing tendency to, 110
causation, 106, 110, 111, 112
cause of death from, 107
citrates in treatment of, 112, 113
conditions arising from, 104
conditions favouring the occurrence of, 109
diet in, 112
drug administration, 113
incidence, 105
intestinal perforation and appendicitis complicating, 111
late effects, 107
pathology of, 108
percentage in series of inoculated and non-inoculated cases, 105
phlebitis preceding, 109
preference for lower limb, 105
prevention of, 112
relative frequency of, 105
rest in bed essential in, 114
symptoms, 110, 111
time of onset, 106, 113
treatment, 112, 113
where the circulation is feeble and blood coagulable, 103
Vocal cords,
paralysis in paratyphoid B, 134
Vomiting,
sign of intestinal complication, 59

War,
first evidence of typhoid fever in, 19
Widal reaction,
possibilities and employment of, 48
Willis, Thomas,
diabetes mellitus first recognised by, 19
early account of typhoid fever by, 19
(*portrait*), 18
Woodville, Wm.,
reference to, on statue of Jenner, 57

THE LIBRARY
UNIVERSITY OF CALIFORNIA
San Francisco Medical Center

THIS BOOK IS DUE ON THE LAST DATE STAMPED BELOW

Books not returned on time are subject to fines according to the Library Lending Code.

Books not in demand may be renewed if application is made before expiration of loan period.

14 DAY

OCT 19 1963

RETURNED

OCT 5 1963

30m-10,'61(C3941s4)4128

82550